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MAYOR.

PHILADELPHIA General HOSPITAL

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AND

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CHIEF RESIDENT PHYSICIAN.

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PREFACE.

It is with great pleasure that the editors present this, the fourth volume of the PHILADELPHIA HOSPITAL REPORTS. The three preceding volumes, the first two edited by Dr. Charles K. Mills, and the third by Dr. George E. de Schweinitz, have been acknowledged successes, both scientifically and historically—scientifically, on account of the valuable contribution to medical science that has appeared in them, and historically, for in them much information relating to the Philadelphia Hospital and Almshouse has been preserved that otherwise would have been lost to the general public and the future historian of this noted institution. The first object of these volumes was to preserve these valuable records, and secondly, to help develop the valuable medical library of this hospital by exchanges from other hospitals throughout the world.

The members of the Staff responded to the call for material most cheerfully and heartily, for which, and many other courtesies, the editors extend their most grateful thanks.

ROLAND G. CURTIN, M.D.,
DANIEL E. HUGHES, M.D.,
Editors.

CONTENTS.

ILLUSTRATIONS.

ADDRESS DELIVERED AT THE OPENING OF THE NEW CLINICAL LECTURE ROOM OF THE PHILADELPHIA HOSPITAL.[1]

BY J. L. LUDLOW, M.D.,

ONE OF THE MEDICAL BOARD AND LECTURER ON CLINICAL MEDICINE.

Extract from the minutes of the Managers of the Philadelphia Hospital:

Resolved, That Dr. J. L. Ludlow be respectfully requested to furnish for publication a copy of the lecture delivered by him on Saturday, the 19th inst., on the occasion of opening the new Clinic Room.

GENTLEMEN: I appear before you to-day by appointment of the Medical Board of this institution to inaugurate the present course of clinical instruction, and to formally dedicate this edifice for the purpose for which it has been erected, through the liberality of the Councils of our city, at the urgent solicitation and representation of our present enlightened and efficient Board of Managers. I hoped that this duty might have devolved upon another, but, situated as I am, I will throw myself upon your generosity and patience while I occupy a short time in giving you a general account of the rise and progress of clinical instruction, the importance with which it has been invested, and to offer such remarks as the present occasion suggests.

It is not my intention to carry you far back in the depths of antiquity and give you a history of medicine. This has so often afforded a topic for introductory lectures, and has been so fully dwelt upon in detail by authors of eminence and learning, that I could add nothing by the incomplete account which I would be compelled to give you at the present time.

That, coeval with the fall of man, disease must have existed and have been disseminated, none can doubt; but how it was alleviated or cured, by what means of a medical character the diseases then existing were managed, we are left in ignorance and conjecture. All the

[1] Delivered Saturday, September 19, 1861.

information we possess is, that (as at later times, though still remote) to the priests in the temples were confided the care of the bodies, as well as the souls, of their fellow-mortals.

The word Clinique, as you are all probably aware, is derived from the Greek Κλίνη, a couch or bed; and by all the term clinique designates that important branch of medical instruction obtained at the bedside of the sick and the information acquired by observation of the actual diseased condition of the patient, with such remarks upon that condition, by a proper teacher, as the occasion suggests.

If we limit the term clinique merely to the study of morbid affections at the bedside, its institution is as old as medicine itself; but if we apply the term to regular and systematic hospital instruction, by professors appointed for that purpose, we look to the second half of the seventeenth century.

It may not be unprofitable for us, gentlemen, to cast a glance back to the more remote ages to learn what kind of cliniques existed in those times. When ignorance and superstition ruled the minds of men, and credulity and fable were the characteristics of the age, we cannot suppose that our science would have shared more than others in enlightenment and true philosophy.

No; though disease stalked abroad and man saw his fellow-man writhing under the torture of pain, or parched by feverish heat or covered with the pestilential leprosy—though the sightless eyeball rolled in its socket, as if looking for some one to destroy the cataract which obscured the vision, or some hideous deformity was intruded upon the passer-by—yet man, selfish though he is, thought little how he might alleviate the sufferings of his race by examining the *structure* of the human frame and the uses of its parts.

We, at this day, wonder why, instead of placing their sick in temples and public places, to beg of the traveller a remedy for diseases and deformities, they should not have endeavored to so investigate the nature of maladies and so apply remedies as to relieve, if not to cure.

Buildings for the purpose of worship were the principal depots for the afflicted. In the temples of Esculapius, however, we find more particular attention paid to the ills of the race. The priests, it is true, dealt more in mysteries and in charms than in clinical instruction; but, as time elapsed, by the observations made constantly upon the

diseased who frequented the temples, something like a science was commenced.

From the Asclepiades, from the master to his scholars, from father to son, through a long line of descendants, the observations in these temples were transmitted until we find them culminating, by intellectual and hereditary right, in the sage of Cos—the immortal Hippocrates—the Father of Medicine.

To Hippocrates we are indebted for those recondite and choice observations in medicine, gleaned and garnered at the bedside, which have crowned him with an immortal wreath and transmitted his name and works to this late day as wonders of research and philosophy.

Had the precepts of Hippocrates been followed, and observation at the bedside been made the basis of practical medicine, instead of vagaries, loose speculations, and hypotheses, the science of medicine would, no doubt, have attained greater perfection even at that distant period.

But the minds of those who followed the Father of Medicine appeared to loathe the only true method of instruction in any science. Instead of induction guiding their judgments, they appeared to revel in a love of crude thoughts, upon which they attempted to build systems upon which they might wrangle and wrestle, if for nothing else than to prove their great powers of mystifying and darkening (by words without wisdom) the simplest truths in nature. As man is alike in all ages of the world, they, no doubt, were very like some of our modern lecturers and speechmakers, who deal much in what the Latin poet calls *Vox et preterea nihil*—a fine speech without matter.

With all due deference to the hoary-headed sires of those ages, I must confess that, while I wonder at the industry which amassed their huge tomes, I am amazed that they could write so much and know so little.

As we descend the stream of time, passing, as we necessarily must, many important names and eras, we are arrested by the name and fame of Galen. Not, however, that he did anything for the advancement of clinical medicine, but that he, by the food which he administered by his writings, disquisitions, and text-books, rather fostered the spirit of controversy which impeded the progress of true observation into the causes of disease for many ages. If clinical instruction at

this time was followed at all, it could only have been by a few in private practice.

It has been asserted by historians that hospitals do not date earlier than the close of the third or beginning of the fourth century. Upon this, however, there remains a doubt. At any rate, cotemporary with the creation of hospitals we must notice the celebrated schools of Alexandria and that of Dschondisabour, in Persia. These schools being in close proximity to the hospitals, the inference is not too strong that they were made use of as part of medical instruction.

Among the earlier hospital schools that of Alexandria, in Egypt, was so celebrated that it is said an assiduous attention upon it conferred the right of practicing medicine.

Previously to this period hospitals had been more particularly appropriated to the care of the sick, but from this time they gradually fulfilled a twofold purpose—that before mentioned and also as schools of medical observation and instruction. When the School of Bagdad was founded by the command of Almanzor it was filled with superintendents from Dschondizabour, where an infirmary had been founded, superior even to that of Alexandria.

As the Arabs proceeded in their conquests, we find hospitals and medical schools following in their wake. Under the protection of that race (the Saracen Moors, who overrun Spain) universities and hospitals sprung up in the cities of Seville, Toledo, and Cordova. At such hospitals Ali-Abbas (a noted Arabian) advised as a great duty the attendance upon the hospital schools, there to observe how little the real appearance and characteristics of disease coincided with the description in the books of the times. Although at this time the advantages from hospital schools appeared to offer every opportunity for the only proper advance in medical science—namely, by observation, close and critical, at the bedside—nevertheless, it really appears as if they wished to blot out the sun and substitute the taper for a luminary in advancing true medicine. Fruitless and vapid discussions assumed the place of strict observation and induction, and disgust drove several to travelling in Italy and France, whence the tide of literature had been flowing. There, too, however, the love of writing and declaiming on medicine, instead of observation, existed. But the number of hospitals increased, and we would have supposed that these would have stimulated proper medical culture. Not so,

however. Writing and speculation with a mass of crude controversy still clouded the minds of the doctors of those times. Revelling in their favorite schemes and dogmas, they almost extinguished the little light which had dawned in the medical world. They most truly forgot the motto of the illustrious Hippocrates: *Ars longa—vita brevis est.*

In proceeding, gentlemen, I think it is not inappropriate to the present occasion to stop a few moments and pay our tribute of veneration and gratitude to two immortal names—Sir Francis Bacon and Thomas Sydenham, the former the father of inductive philosophy, the latter the restorer of Hippocratic medicine; the former the pioneer in correct reasoning, the latter the applier of that system to the development of our science. The influence of the Baconian philosophy upon Sydenham we may learn from the following passages:

"As truly as the physician may collect points of diagnosis from the minutest circumstances of the disease, so truly may he also elicit indications in the way of therapeutics. So much does this statement hold good that I have often thought that, provided with a thorough insight into the history of any disease whatsoever, I could invariably apply an equivalent remedy—a clear path being thus marked for me by the different phenomena of the complaint. These phenomena, if carefully collated with each other, lead, as it were, by the hand, to those palpable indications of treatment which are drawn, not from hallucinations of our fancy but from the innermost penetration of nature.

"By this ladder and by this scaffold, did Hippocrates ascend the lofty sphere. The Romulus of Medicine, whose heaven was the empyrean of his art, he it is whom we can never duly praise. Herein consisted the theory of the divine old man. It exhibited the legitimate operations of nature put forth in the diseases of humanity. The vain efforts of a wild fancy, the dreams of a sick man, it did not exhibit."

I could quote further, with advantage to us all, but I must hasten onward.

Passing now to the middle of the seventeenth century, our attention is arrested by the establishment of cliniques in Holland. Yes, gentlemen, to Holland—that land more maligned and calumniated than any country in Europe, and yet a land around which more glorious recollections cluster than any land in the Eastern Hemisphere; a spot which suffered more for civil and religious liberty than almost any part of the world—the country of William and Mary. The country

which gave William and Mary to England, and by them the Toleration Act. The home of the oppressed, the refuge of the Puritans before they set sail across the broad Atlantic for New England's rocky coast. The land, which when given a choice between money and a university, chose a university. A land of literature and letters, of science and art. A people who rescued their country from the sea. A land of dykes and canals, and a people who, when they had not land enough, pumped out a lake. And yet they call this people, with a sneer, phlegmatic Dutchmen. I cannot tell whence came the delusion, excepting that they, with all their industry, prowess and literature, knew how to enjoy life. The old men with their long pipes and the venerable dames with their huge caps and their well stored larders, which they knew how to prepare, with easy consciences and a sufficient rotundity of body to keep them warm in winter and perhaps a good glass of Holland gin for a night-cap. If you doubt what I have said, read Schiller and Davies and Motley and Brodhead and Prescott, and I know you will bear me out in my assertions. But to return.

In 1643, clinical establishments flourished in Holland. The most distinguished was that at Utrecht, under Straeten, and at Leyden, under Huernius. He was the first to teach bedside medicine upon a comprehensive scale. After examining closely the patient in the presence of his pupils he expatiated upon the nature of the diseases and also frequently caused post-mortem examinations to be made.

In addition to Huernius we have DeLaboe, who was the first to pnblish a connected series of bedside observations with remarks, and this system was pursued by his successors. To DeLaboe, of Leyden, we are indebted for the first clinical reports. The next great clinical teacher is the illustrious Boerhave. Under his instruction some of the most distinguished physicians of Europe were educated who scattered throughout the land the fruits of the good seed which had been sown by Boerhave, at Leyden.

Now we find the spirit of proper medical instruction, based upon clinical investigation, spreading to Scotland and afterwards to Vienna, whose schools soon eclipsed the mother institution.

In Scotland, Dr. Rutherford, in 1748, commenced his course of cliniques in the Royal Infirmary in Edinburgh, in which he was soon joined by other professors in 1757. I have in vain endeavored to ascertain when cliniques were introduced into England and Ireland, where

we know they at present exist in great perfection. I gather, however, from the histories of some of the older hospitals, that the hospital of St. Bartholomew was instituted in connection with the priory of St. Bartholomew, in 1122, and after undergoing various modifications in its charter (in which Christ's Hospital, Bridewell and St. Thomas's were included) it became more perfectly established in 1752.

Yet, from a historical oration delivered at the opening of one of her sessions of lectures, I find that it was not before 1822 that John Abernethy, founded the present system of lecturing, though Percival Pitt delivered occasional lectures some eighty years ago. St. Thomas's Hospital was founded in 1213, and after being subjected to many changes, was finally well endowed in 1872, to which a medical school was attached but the exact date I cannot give.

Guy's Hospital was founded by Thomas Guy, of London, in 1725, and in 1760 an arrangement was made within St. Thomas's Hospital, by which the privileges of both hospitals were extended to the pupils of each. In Dublin, the Meathe and Sir Patrick Dunn's Hospitals and others have been open for years for clinical instruction, but when they were opened I have not been able to ascertain.

Five years after the cliniques had been originated by Dr. Rutherford, in Edinburgh, a clinical institution was established at Vienna by Boerhave, where lectures were delivered by Van Swieten, who was succeeded by DeHaen, who gave the results of his observations and experience to the public, supported at times by necrological examinations in his *Ratio Medendi*, a work of sixteen small volumes, in which he insisted upon the Hippocratic method of investigating disease. DeHaen was followed by Stoll, who gave a still greater impulse to the Viennese clinique.

While the clinique was thus progressing in Vienna, we now find Dr. Cullen giving clinical lectures in the Royal Infirmary of Edinburgh, assisted by Drs. Wyeth and Moore, and afterwards Gregory and Home.

The combined influence of the good effects of cliniques as subservient to high-toned medical instruction, in Holland, Vienna, and Scotland, excited at this time a general interest in clinical study in other schools of Europe—in Gottingen and Sarthe, etc. In Italy, in 1715, a clinique was established by the Pope, in connection with the hospital at Rome, amid all the pomp and splendor of the church, in presence

of Cardinals and Prelates, with orisons and vespers, and the full diapason of the multitude. Lancisci was inaugurated as Chief of the Clinique in the vast Hospital of the Holy Ghost. In passing, I may remark that we find a species of clinique attached to the Hospital of St. Francis, in Padua, as far back as 1578, but of this we know very little; it probably, from all we can gather in regard to it, did not deserve the name, even in the most restricted sense.

In the University of Pavia, in 1781, Tissot and Scarpa occupied clinical chairs; while at Bologna, Tommisini was occupied, and at Milan, Rasori.

In Spain, also, we find cliniques established, and pupils are compelled to see a town practice of two years, before entering upon private duties.

In Russia, clinical instruction has been established since 1765, and continued on a most extensive and vigorous scale. The more proficient clinists, to a certain number, are sent at the expense of the government to other countries, to gather what is new in medical and surgical knowledge. In Prussia, it is said, cliniques are more insisted upon than even in Russia.

In the cursory glance which I have given you, gentlemen, of clinical establishments, I have designedly said nothing of France. That country deserves especial commendation, for, since their inception, the attention which had been paid to clinical instruction has vastly surpassed, as a whole, that of any other country on the globe. Previous to 1786, when reforms were introduced into the University of Caen, and a chair of practical medicine was attached to the Hotel Dieu, DeBois, Physician to LaCharité, gave clinical lectures to a limited number of pupils.

During the convulsions of the state which lacerated France at the close of the eighteenth century, medicine alone remained stationary. Her votaries, alive to the interests of humanity, were ceaseless in their efforts to advance medical science, and during every lull in the political tempest, the subject was pressed upon the attention of the government.

In the year 1794, cliniques became attached directly to the faculties of medicine, and simultaneously the schools were provided with clinical professors. Desault occupied a chair of clinical surgery, and at the death of DeBois, Corvisart was appointed Physician to LaCharité, and in 1795 professor of clinical medicine.

Corvisart being selected as medical adviser to the Emperor Napoleon, on the first day of the consulate used that influence with the first consul which his position and talents afforded him, and which might have been expected from one so distinguished in the annals of medicine. Among the pupils of Corvisart we find Bayle Lannec and Baron Dupuytren. Pathology engaged his attention. Percussion, and investigations upon the diseases of the heart were illustrated at the bedside of the patient. During the professorship of Corvisart, many of the most eminent practitioners of succeeding years were educated, and thence dispersed throughout various parts of the empire. At this time more attention was paid to pathological anatomy, and post-mortem examinations were extensively and vigorously carried out. In addition to the cliniques in Paris, Fouque founded clinical instruction in the old and time honored University of Montpelier, in which he was ably seconded by Delpech. At Strasburg, after a great length of time, and much persuasion, an obsetrical clinique was founded.

Though France has been late in entering the clinical field, yet she soon made up by her efficiency what she had lost by her tardiness.

The multitude of her clinical institutions are unrivalled. There is not a city in the world where equally numerous and varied clinical courses are delivered as in Paris.

The science of practical medicine owes much to the energy and industry of our Galic brethren. Nowhere has there been so close an analysis of disease and so scrutinizing investigation.

In later years the discoveries of the teachers of Germany, Austria, England, Ireland and America, have added much to our previous knowledge of physiology, pathology and the general science of medicine. But for a long period before, France must be acknowledged to have done more than any other country, and all this may be attributed to the spirit of research suggested by bedside observation and clinical lectures.

I have now, gentlemen, though with rapid strides, endeavored to give you an account of the rise and progress of clinical instruction in the old world, touching, to be sure, but lightly many important points, which of themselves would afford ample scope for a lecture. You have seen the importance attached to bedside instruction in the Eastern

Hemisphere, and the fact that medicine only became rational and philosophical when founded upon the observations made at the couch of the sick.

I now will bid adieu to our trans-Atlantic brethren, to give you a history of the origin of clinical medicine on our continent.

Notwithstanding the American Colonies boasted several medical characters of note, and as early as 1750, the body of a criminal, Harmanus Carroll, executed for murder, was dissected, and the blood-vessels injected for the instruction of medical students, by Drs. John Bard and Peter Middleton, of New York. (The first recorded dissection of the human body for medical purposes in this country.) And although in our own city lectures had been delivered, yet no systematic course of medical instruction was given in this country until 1765, when Drs. Shippen and Morgan delivered the first course of lectures in this city.

The following advertisement relating to the opening of the medical school in the new world may not be uninteresting to you, and I therefore will repeat it: "As the necessity of cultivating medical knowledge in America is allowed by all, it is with pleasure we inform the public that courses of lectures on two of the most important branches of that useful science, viz., anatomy and materia medica, will be delivered this winter in Philadelphia. We have great reason, therefore, to hope that gentlemen of the faculty will encourage the design, by recommending it to their pupils, that pupils themselves will be glad of such an opportunity of improvement, and that the public will think it an object worthy of their attention and patronage. In order to render the courses more extensively useful, we intend to introduce into them as much of the theory and practice of physic, of pharmacy, chemistry and surgery, as can conveniently be admitted. From all this, together with an attendance on the practice of the physicians and surgeons of the Pennsylvania Hospital, the students will be able to prosecute their studies with such advantage as will qualify them to practice hereafter with more satisfaction to themselves and benefit to the community." Then follow the time, terms, etc.

Dr. Morgan was appointed professor on the 3d of May, 1775, and Dr. Shippen on the 17th of September of the same year. To Dr. Morgan, therefore, we are indebted as the founder of the school of Philadelphia. After this Drs. Rush and Kuhn were added to the med-

ical faculty of the College of Philadelphia, afterwards the University of Pennsylvania.

In the first introductory lecture delivered in this city and upon this continent, by Dr. John Morgan, in 1765, the following among many other cogent reasons for establishing a medical school in Philadelphia is educed, and one to which he, just from the influence of bedside instruction in Europe, attached the greatest importance. I quote from his lecture : " Besides men of great abilities and eminence, under whom they may see private practice, the hospital of this city is a great persuasion to determine a concourse of medical students to this place. It would be doubly useful to them, and increase their motive of repairing here, if they could be first properly initiated in the principles of their profession by regular courses of lectures, duly delivered. This noble charity (referring to the Pennsylvania Hospital), maintained by the donations of the benevolent, at no small expense, supports a great number of sick of both sexes, as there are six physicians of the most unquestionable skill, integrity and character, who, moved by the tender principles of humanity, have taken upon them the charge of attending the sick gratuitously. Pupils here meet with such a number of cases, both chronic and acute, treated so judiciously and so agreeably to the rules of art, as cannot fail very much to faciliate the knowledge of their profession." He then goes on to remark, that since, of the six physicians to the hospital, five were members of the Board of Trustees of the College, he might infer that everything in their power may be hoped for to second the medical institution of the college by uniting with them the advantages of the hospital, and thus render the education of youth in the healing art as complete as possible in this city; to which nothing can contribute more, than a course of clinical practice and clinical lectures.

And now, gentlemen, we have arrived at that epoch in the history of medicine in our country, when the first clinical lecture was delivered in America. In the Pennsylvania Hospital, on the 3d of December, 1760, Dr. Thomas Bond, one of the physicians to that institution, gave the introductory clinical discourse.

I cannot forbear quoting from this lecture, if not for instruction, nevertheless from curiosity, that we at this late day may see how the father of clinical instruction in America appreciated the advantages which must necessarily arise from a proper clinical course.

Speaking of Dr. Morgan, the Professor of the Theory and Practice in the University, he remarks: "The field this gentleman undertakes is very extensive, and has many difficulties, which may mislead the footsteps of an uncautional traveller. Therefore, lectures in which the different parts of the theory and practice of physics are judiciously classed and systematically explained will prevent many perplexities the student would otherwise be embarrassed with, will unfold the doors of knowledge, and will be of great use in directing and abridging his future studies. Yet there is something further wanting: he must join examples with study before he can be sufficiently qualified to prescribe for the sick, for language and books alone can never give him adequate ideas of diseases and the best method of treating them; for which reasons infirmaries are justly reputed the grand theatres of medical knowledge. There the clinical professor comes to the aid of speculation, and demonstrates the truth of theory by facts, etc."

Further on again, he says: "I am now to inform you, gentlemen, that the managers and physicians of the Pennsylvania Hospital, on seeing the great number of you attending the School of Physic in this city, are of opinion that this excellent institution affords a favorable opportunity of further improvement to you in the practical part of your profession, and, being desirous it should answer all the good purposes intended by the generous contributors to it, have allotted to me the task of giving a course of clinical and meteorological lectures in it, which I cheerfully undertake."

Thus, gentlemen, in a small beginning, but a beginning upon the right principles of studying medicine, as founded in the City of Philadelphia, has been erected that medical character, reputation, and renown which has eclipsed all the other cities on our continent, and made Philadelphia (which may she long continue to be) the metropolis of medical science in the Western Hemisphere.

The example of Philadelphia, in instituting a medical school, was followed by New York in 1768, and in 1769 the New York Hospital was started, but was not finally completed until 1791; and one of the chief inducements to its erection was to aid the School of Medicine, by disseminating proper clinical instruction. Since the days of the venerable Bond, an enlightened and comprehensive policy has prevaded the succession of managers of the Pennsylvania Hospital; and the wards of that institution, with corps of teachers ably qualified, have

furnished some of the best investigations in this or any other country. In speaking of hospitals, I would not detract one iota from their general management throughout our country, for in almost every other place where a hospital has been established it has been made subservient to medical instruction.

Near the commencement of the present century the wards of the old Philadelphia Almshouse and Infirmary were opened for clinical lectures, and continued so during the time that institution was within the city limits, on the square between Spruce and Pine and Tenth and Eleventh Streets. At that time, the classes being limited in number, the students followed the attending physician and surgeon in his daily walks through the wards, in imitation of the European plan; but as the classes grew larger, particularly in the winter seasons, on account of the celebrity of our medical school, it became necessary to initiate the present plan, which was suggested by Drs. Chapman and Jackson, then attending physicians. The Board of Managers, at that early day, seconded their efforts, and had constructed clinical wards, and a lecture room built, where patients were introduced as at present, and their cases investigated, lectured upon, and treated.

It may not be irrelevant to remark, in passing, that at that period the attending physicians and surgeons received for their services each fifty dollars a year, and the sum of ten dollars was demanded for the ticket entitling the student to attend the clinical course. As the managers had built a special lecture room for the benefit of the students, the Medical Board relinquished all claim to compensation for their services, provided the board would appropriate this sum, together with the fees from the students, to the formation of a library fund. This was done, and most of the books at present in our library were accordingly purchased. The fund in a few years amounted to several thousand dollars, and was gradually increasing, and important medical and surgical works added; but as the sum became larger, and books were not purchased with that rapidity with which modern bibliomaniacs fill their libraries, and often spend fortunes for comparatively worthless volumes, and a new board coming into office, all the accumulated library fund, by one fell swoop, was thrown into the common hospital fund, and, as a matter of course, the place that knew it once knew it no more forever. I am happy to say, however, that the gentlemen composing our present board, in addition to the numerous

and salutary reforms which they have introduced in this institution, deserve the thanks of our profession for the consideration which has once more dictated an annual appropriation for our library, and also for the establishment and preservation of the Pathological Museum. When, by the march of improvement, it became necessary to remove to more extended and distant quarters, and these buildings were erected, the wards still continued accessible for clinical investigations, and the spacious amphitheatre we have just vacated was constructed to accommodate the large classes which annually were brought to our city by the reputation of our medical colleges.

I only wish, gentlemen, that I could say that they have always remained open to diffuse the benefits to humanity which always must accompany a well-regulated hospital. But other counsels prevailed; misguided philanthropy, with reasons which most judicious men could not appreciate, closed these wards to all clinical instruction, and for nearly ten years they remained so. When by an Act of Assembly of the Commonwealth of Pennsylvania the City and Liberties became consolidated, and a new Board of Guardians were elected, among the first of its acts was the opening of this house again. Since then the hospital, with the exception of one year, has been available to students visiting our city.

The cost of supporting this institution, according to the Annual Report of the Mayor to the Councils of our city, on the 31st of January, 1861, was $186,398.75.

Within these extensive walls, in the hospital proper, during the year up to January 31, 1861, more than 8000 patients were treated in the medical and surgical wards, and we have nearly 800 beds almost always full. During the last three months there has been under treatment, in the men's medical wards alone, 409 patients with almost every variety of disease. In giving the above statistics we have not taken into account the insane asylums. These are separate apartments, and, I may add, that out of a population at any one time averaging from 2400 to 2600 individuals, excluding, of course, the children, we have not in the house from forty to fifty able-bodied persons. This may give you some idea of the facilities of this institution in affording subjects for clinical investigation.

Philadelphia owes much of her celebrity to her medical colleges. She has been aptly termed the Emporium of Medical Sciences in the

Western World. The gentlemen who have filled the important posts of physicians and surgeons to her hospitals have shed lustre upon her.

The present Board of Guardians of this institution, representing the City of Philadelphia, imbued with the proper spirit of humanity, and anxious to subserve the interests of medical science, have, with a laudable spirit, invited you to avail yourselves, without fee, of its advantages, and have erected this building in close proximity to the hospital in order that acute and important cases of disease can be brought before you without danger to the patients. It remains with you how you will respond to these opportunities for bedside instruction.

Besides all this, by the advantages we possess for necrological examinations, our institution is unsurpassed by any in the country. Here, dealing with the living and the dead, there is scarcely a point in diagnosis or pathology which we cannot illustrate.

To render this lecture more complete, it would have afforded me great satisfaction to have dwelt upon the various modes of clinical instruction pursued in past times, and practiced at present in the various hospitals in the world, but I must forbear. I have already, I am afraid, wearied you. But I cannot, however, close this lecture without pressing upon you, by the examples of the past, and the hopes for your future, the necessity of close clinical study. You may learn from the professors in the schools the theory of medicine and surgery, but only at the bedside of the patient can you be taught the intricacies of practice.

You, gentlemen, have chosen a noble, honorable and learned profession—full of responsibility, toil and trials. The inducements to follow it lie within itself and the good of humanity.

The course of the physician to honor and distinction, is not in the bustle and strife of the world, but in that still, quiet round of duty, which, like our blessed Master, was in going about doing good.

The conqueror's wreath is not woven for you, although you may be more than heroes, standing in the breech when pestilence and death are hurrying thousands to their graves.

Listening Senates will not hang in admiring wonder upon your sentences, though by your counsels you may warn a nation from an impending disease, which will tap the life-blood of the people.

No, gentlemen—by the bedside of the sick, doing battle with the King of Terrors, warding off the arrows of the insatiate archer, and

pouring the oil of gladness in the sick man's bosom—that will be your battle-field—that your forum. Or perchance the agony of your nation, travailing for existence, may call you from the peaceful pursuit of your profession in civil life to the military camp or the bloody field of war. Here, too, no reward such as wreaths the victor's brow awaits you, though, by your skill and attention, disease more fatal than all the accidents of battle may be averted or mitigated—or in the bloody fray, when the wounded are covering the field, you, in moral grandeur, go not forth to mark your track by blood, but, regardless of self, seek to bind up the wounds of the soldier, or, by your godlike skill, save hundreds from death.

Some of you may become professors in the various schools of our country, but that number must necessarily be limited. If so, your responsibilities are increased, for if you possess nothing else, the very position you occupy gives you prominence, and the lectures you may deliver may be of vast benefit, or of incalculable evil, for from those schools as centers, hundreds will go forth either for the healing or the death of the nation.

How to acquire this god-like profession, is a subject of vast moment to you.

System with you is time. Many a noble fellow has entered upon the study of medicine full of ardor and with high hopes. He was anxious to do his best. After reading a short time in the office of his preceptor, he hies away to attend lectures. Before leaving home he has determined to be a doctor in two years, and in some cases less than one. To effect this he must attend all the lectures in the school, he must dissect, or rather he must cut up a dead body in the dissecting room, for most students know very little about the proper principles of dissections; he must take a quizer's ticket, but as for attending clinical instruction, excepting the operations in the lecture room of the college, or listening to the stories of some peripatetic patients, with the few remarks of the professors upon the cases, he has not time for them. No, no! He studies for a diploma. Study for a diploma! A paltry piece of parchment two feet by two with a piece of blue ribbon and a big lump of sealing-wax attached, covered with a copper-plate inscription, all in Latin, which, possibly, he may be able to translate, but which most probably he cannot, taken from the back of a sheep, and sometimes given to a sheep. Why, gentlemen, this is not study-

ing medicine, I know not what to call it, excepting an approved course to become mental and physical dyspeptics.

Studying for a diploma! No, gentlemen, let not this be said of you; take a sufficient time and systematize your studies. Do not cram all into two, but extend it over three years at least, and then do not merely remain here during the winter course, but also during the spring and fall. You will find able and willing hands in our city to direct your studies, and assist you in prosecuting many of the most important branches of scientific and practical medicine, and above all attend clinical instruction in our hospitals and dispensaries.

This will be studying medicine and surgery to some purpose.

And when you come forward to assume the Doctorate, you will not come before your professor almost cringingly, with nervous tremors over your body and your heart beating as if anxious to leap from your breast, and the cold sweat drops upon your brow, and your hand as cold as icy marble, as if begging for your diploma. No, but with a sparkling eye and glowing face and head erect, conscious of your medical manhood, you will court the most scrutinizing examination, and by right of your requirements, demand your diploma.

You will honor the diploma, and not the diploma honor you.

The school from which you graduate, and the professors who have taught you, will point to you then as their jewel.

Time warns me to conclude. The subject which I have attempted to present to you to-day, I am better aware than you are, has not been treated with that fullness and completeness which its importance demands. The space of time allotted to me necessarily imposed brevity.

But, gentlemen, if I have by the history of the past, and the magnitude of the theme, aroused you to the necessity of attending clinical investigations, my object will be attained.

MEMORIAL ADDRESS UPON THE DEATH OF ALFRED STILLÉ, M.D., LL.D.[1]

BY ROLAND G. CURTIN, M.D.

It is with sadness that I accede to the request of the Chairman of the Executive Committee to speak briefly of our late President, Dr. Alfred Stillé, who has passed away since our last meeting. He was greatly interested in this society and cordially supported our efforts in the early stages of its formation, and continued to meet with us as long as his failing health permitted. He has been our president since our organization. Upon one occasion he read a valuable historical paper before this Association, entitled, "Reminiscences of the Philadelphia Hospital," which was afterward published in the first volume of the PHILADELPHIA HOSPITAL REPORTS.

Dr. Stillé was my professional father in the early walks of medicine, first as professor in the medical department of the University of Pennsylvania, and later as visiting chief in the medical wards at Blockley. Those who enjoyed his courteous encouragement when he reviewed their work will remember his kindly approving manner, and likewise those who failed in their duty will always have before them his disapproving glance, one of which was enough.

Dr. Stillé was a descendant of Olaf Stillé, one of the early Swedish settlers who came up the Delaware in 1638. Dr. Stillé was born in Philadelphia, October 30, 1813, received his academic education in his native city and entered Yale College in 1828, and after completing his sophomore year came to the University of Pennsylvania, entered the junior class and graduated in 1832. He then entered the medical department of the University of Pennsylvania and graduated in medicine in 1836. In the same year he entered Blockley as resident physician, and after his term ended he went to Paris and studied two and a half years. Returning from Paris he entered the Pennsylvania

[1] Read before the Association of Ex-Resident Physicians of the Hospital at the annual meeting, December 4, 1900.

Hospital and served two years as resident physician. He began the practice of medicine in Philadelphia, in 1845, and commenced his career as a lecturer in the Philadelphia Association for Medical Instruction, continuing until 1851.

Dr. Stillé was a visiting physician and lecturer to the Philadelphia Hospital from 1865 to 1871. He also served as visiting physician to St. Joseph's Hospital from 1849 to 1877. He was professor of practice of medicine in the Pennsylvania Medical College from 1854 to 1859, and in the medical department of the University of Pennsylvania from 1864 to 1884. He served with great faithfulness and renowned ability.

During the great Rebellion, Dr. Stillé served as an army surgeon in the Satterlee Hospital in West Philadelphia from 1862 to 1863.

Dr. Stillé left the University of Pennsylvania his large and valuable medical library, which has increased to magnificent proportions. He was a writer of great force and accuracy, being classical and very precise. He left valuable works on epidemic cholera and epidemic cerebo-spinal menigitis, the result of his observations in the wards of Blockley. He translated many valuable French essays relating to medicine, and with Professor Maisch edited the National Dispensatory.

After this short review of a portion of the valuable work of Dr. Stillé let us pause a moment in our joyousness to think of the clean character he left to us, one worthy of emulation by his followers in Blockley. Peace to the ashes of our departed president. Farewell, a sad word, but it seems doubly so when we apply it to a colleague so cultured and so useful and sweet as Dr. Stillé.

STATISTICAL TABLES OF THE GENERAL AND INSANE HOSPITALS.[1]

By D. E. Hughes, M.D.

General Hospital.

There are remaining in the wards to-day 1084 patients, 3 less than the number remaining December 31, 1898.

The number of admissions during this year was 6956, and adding to the 1087 remaining December 31, 1898, gives a total of 8043 patients who have received medical care and treatment during the year, a gain of 28 over the number treated during 1898.

The average daily population has been 986.

The condition of the 5903 patients at the time of discharge was: 3229 cured, or nearly so; 1621 improved, and 983 unimproved.

Of those discharged unimproved, 609 were admitted to the insane wards upon legal commitments. Of the patients discharged from the hospital wards, 570 (423 men and 147 women) were transferred to the out-wards.

The number of patients dying has been 1056, a mortality of 13 per cent., which, while heavy, is not surprising when the character of the illness, its duration and the depraved condition of those admitted is understood.

Four hundred and forty-two men and 358 women, suffering from acute and chronic mental ailments, were admitted to the detention wards during the year. This is the only general hospital in this city receiving mental patients, and as a consequence many patients are sent here as a matter of public and personal safety pending the consideration for their final disposition. Mental cases, accompanied with the proper legal commitment papers, are not admitted direct to the insane hospital, but are sent with those without commitment papers to the detention wards pending an inquiry into the char-

[1] Extract from the Report of Dr. D. E. Hughes, Chief Resident Physician of the Philadelphia Hospital (General and Insane), for the year 1899.

acter of their mental diseases. In a fair number of instances very rapid recovery results in sudden mental outbreaks, and the patients are, in a few days or weeks, discharged from the hospital, in this way reducing considerably the admissions to the insane hospital, whose wards are badly overcrowded.

Seven hundred and forty-six men and 86 women have been cared for in the alcoholic wards. Of this immense number a large proportion are admitted several times during the year. There is no denying the fact that very many of the cases of alcoholism and drug habit sent to the hospital are not of that severity calling for hospital care, and results in the alcoholic wards being continually overcrowded. It is considered more human, however, to accept all these cases sent to the hospital rather than by some unfortunate error refuse one whose alcoholic symptoms are apparently trivial and death follows his refusal, as there is nowhere else for the self-destroying individual to go. The victims of the alcoholic and drug habit are of low resisting power physically, and from undue exposure and prolonged abstinence from food are an easy prey to unforseen accidents and disease.

The wards set aside for patients with pulmonary consumption have been crowed during nearly the entire year. The men's wards cared for 413 patients (many of these second and third admissions) and the women's wards 118. These wards were set aside for this class of patients four years ago, and a regulation made that the moment a patient in any ward of the hospital has had the diagnosis of tuberculosis made, transfer to the phthisis wards must at once follow.

The custom of caring for sick children in adult wards has been continued for the reason that we lack proper children's wards. There is a great need for better facilities for children above the nursery age.

Sixty patients, mostly children, suffering from measles, whooping-cough and diphtheria, have been cared for in the Isolating Pavilion. This building, situated a considerable distance from the hospital buildings, was erected to care for only such cases of contagious diseases as might develop in or soon after admission to the hospital. Its use has, however, been widened by the regulation permitting the admission of cases of measles, whooping-cough and chicken-pox from other institutions and from the homes of those who are unable to provide for them. If this course is to be continued an addition to the pavilion must be considered.

The ambulance service of the hospital is the most thoroughly equipped of any in the city, two modernly constructed ambulances being always in service, with a third in reserve. During the year 1242 calls were made, to accomplish which 7383 miles were travelled.

The facilities for clinical teaching have been continued under the same liberal arrangements as heretofore, and 8900 visits to clinics (in the spacious and fully equipped clinic hall), and in the wards (in classes not exceeding ten), under the immediate supervision of the visiting surgeons and physicians, have been made by medical students.

Two hundred and eleven autopsies have been performed, complete records of which are preserved.

The hospital library contains 3500 volumes. The latest works are added each year for the use of the internes. A large number of medical periodicals are also available for those wishing to keep abreast of the present advances in the art and science of medicine.

SUMMARY OF PATIENTS TREATED, 1899.

WARDS.	Treated.	Cured.	Improved.	Unimproved.	Discharged.	Died.	Remaining.
Men's Medical	1912	614	526	79	1219	442	251
Men's Alcoholic	746	220	68	3	691	23	32
Men's Detention	442	12	10	386	408	21	13
Men's Surgical	763	315	203	35	553	68	142
Men's Venereal	349	138	160	19	317	4	28
Men's Eye	101	50	27	7	84	1	16
Men's Skin	90	39	37	3	79	.....	11
Men's Nervous	479	33	121	72	226	68	185
Women's Medical	790	218	189	31	438	227	125
Women's Alcohol and Detention	444	80	48	286	414	20	10
Women's Surgical . . .	260	89	57	14	160	34	66
Women's Venereal . . .	143	61	63	2	126	4	13
Women's Eye.	63	33	15	2	50	5	8
Women's Skin	46	18	14	2	34	.....	12
Women's Nervous . . .	182	4	35	21	60	40	82
Gynecological	134	67	20	9	96	27	11
Obstetrical and Maternity	293	276	3	2	281	5	7
Nursery—Adults	188	153	12	4	169	1	18
Nursery—Children . . .	397	285	13	6	304	63	30
Children's Department .	221	194	.....	.....	194	3	24
Grand total	8043	3299	1621	983	5903	1056	1084

INSANE HOSPITAL.

The admissions during 1899 to the insane hospital were the largest in the history of the institution, the number being 609—329 men and 280 women, or—11 more than during 1898.

For the ten years ending with this year, there has been admitted to the insane hospital, under legal commitment, 2631 men and 2284 women, a grand total of 4915. The admissions for each one of the past ten years is shown in the following table:

NUMBER OF PATIENTS TREATED DURING THE PAST TEN YEARS, WITH THE ADMISSION EACH YEAR, RESPECTIVELY.

	Men.	Women.	Total.
Patients in wards, January 1, 1890	403	421	824
Patients admitted in 1890	155	128	283
Patients admitted in 1891	150	128	278
Patients admitted in 1892	256	241	497
Patients admitted in 1893	262	204	466
Patients admitted in 1894	258	221	479
Patients admitted in 1895	282	240	522
Patients admitted in 1896	313	278	591
Patients admitted in 1897	321	271	592
Patients admitted in 1898	305	293	598
Patients admitted in 1899	329	280	609
Total	3034	2705	5739

The number of patients receiving care and treatment during the year was 1987, an increase of 62 over the preceding year.

The total population this day is 673 men and 697 women, a grand total of 1370, or 8 less than at the beginning of the year. The average population has been 1382—694 men and 688 women.

The number of patients now under care in the insane hospital is far in excess of the capacity of the wards, and has greatly interfered with the proper classification and individual care and study of the immense variety of mental and physical symptoms presented by the large and growing population. The successful treatment of mental diseases depends, to a very large extent, upon a thorough appreciation of the various hallucinations and delusions of the individual and the ability of the medical staff of the hospital to gain the confidence and respect of the patients. The population has been too large, the wards too crowded and the admissions too great to permit my devoting to each individual the necessary time to insure all the beneficial results that

the mattress shop, have been placed in the wards, replacing the same number of straw beds. It is hoped during the coming year to have all the wards supplied with the new mattresses which have proven so successful since their introduction a year and half ago.

The "rain baths" still coutinue the most efficient and healthful plan of bathing yet employed, permitting the thorough bathing of the entire population several times weekly without any special effort, an impossibility when tubs were used.

The house telephone is an important addition to the hospital equipment.

One of the important needs of this portion of the institution is better accommodations for the attendants. The successful management of an insane hospital depends to a very large degree upon the intelligence, faithfulness, contentment and comfort of the employees. Sleeping rooms on the wards are not conducive to health and comfort. During the year there has always been several attendants away from duty on account of sickness, and generally the causes were the result of their unhygienic rooms.

Closing this, my tenth annual report as Chief Resident Physician of the General and Insane Hospitals, I desire to make mention of the changes and additions made to the duties and responsibilitles of the position since my appointment. On May 31, 1887, the position of Physician-in-Chief of the Insane Hospital was abolished and the insane wards were attached to the nervous wards of the general hospital, thus placing the care of the insane patients under the direction of the visiting neurologists.

The men's insane wards and the male attendants were placed under a supervisor (male) who reported directly to the superintendent. The women's wards were under the management of the chief nurse of the general hospital, and the pupil nurses of the general hospital were on duty in the women's insane wards. The position of the chief resident physician was in reality the medical warden, if such a term could be framed, of the institution. The position was delightfully free from responsibility and worry.

In June, 1890, upon the request of the visiting neurologists, the insane wards were severed from all connection with the nervous wards by the adoption of the following rule: "The hospital for the insane shall be under the medical control and management of the Chief Resident

ten to fifteen years, 40 between fifteen and thirty years, 10 between thirty and thirty-five years, 1 over thirty-six years, 1 over forty-one years and another over forty-eight years.

A study of a table showing the length of residence of such an army of demented and helpless individuals presents the strongest argument for the early consideration of the ways and means to properly house and care for them, as the number are rapidly increasing, and with increasing residence in crowded wards comes increased mental and physical decay, shown so distinctly in the yearly increase in the death rate of the hospital and, worse than all, the interfering with or decreasing the chances of mental recovery of the acute insanities.

During the year the same attention has been given to the useful employment of patients that has been so successful in the past, the average number of men usefully working during the year being 320, and the women 309. The practice of keeping all patients physically able in the open air for hours during clement weather has been continued with the attending beneficial results, thus allowing prolonged ventilation by open doors and windows nearly all day long the year around; and not only are the patients kept in the yards, but for several hours a day great numbers are kept marching or walking in companies under watchful care of attendants. These marching columns of hundreds of insane patients daily witnessed in our yards is the wonder and surprise of visitors. To the beneficial results from prolonged open-air life, active marching for several hours at intervals during the day, and the entire absence of entertainments or excitements of any kind after supper, is to be explained the quiet wards during sleeping hours, which is the rule in this hospital.

It has not been necessary to seclude a single patient during the year, and but two men and five women have been restrained, and these seven individuals only for the periods during which their homicidal delusions were dangerous to those about them. It has long been demonstrated by the management of this hospital that the easiest way to manage insane individuals is to offer them the greatest amount of freedom consistent with safety to themselves and those about them, ever on the alert for the first evidence of disturbance. The greater the restraint of liberty and action the more disturbed and difficult becomes the question of control.

During the year, 940 "coir fibre" mattresses, made by patients in

A CASE OF GALL-STONE IN THE COMMON BILE-DUCT, WITH CHILLS AND INTERMITTENT FEVER (BALL VALVE OBSTRUCTIONS. HEPATIC FEVER).

By JAMES TYSON, M.D.

I am indebted to Dr. Frank S. Bowman, my resident physician during the summer of 1898, for the notes of this interesting case. The patient, a German, was admitted to Philadelphia Hospital, June 19, 1898. He was sixty-eight years old, had been married and was a hostler by occupation.

Personal History.—Born in Germany; has been in America thirty-four years, eight of which have been spent in Baltimore.

Present Illness and Condition on Admission.—He says he has been sick for about one year, the chief trouble being dizziness in his head. This vertigo has been so great that he has fallen in the street several times lately. He has a slight cough. Temperature has a peculiar range—from sub-normal to 101°. He says that he has had fever at times while at home. The radial arteries are hard and tortuous. Pulse slightly irregular and slow. The breathing rate is normal. There is a slight degree of jaundice about the face and body.

Physical Examination.—The apex-beat is palpable but not visible in the fourth interspace one-half inch within the nipple line. The normal sounds, as heard at the mitral and aortic areas, are feeble. There are no murmurs. The cardiac percussion areas correspond with those of health except that the upper border to the left of the sternum seems a little higher than normal.

Vocal and tactile fremitus are increased at the apex of the right lung where there is also slight impairment of resonance, and auscultation also recognizes a breathing almost bronchial in character. It is clear, loud and blowing in quality. It is evident there is arterial sclerosis, with catarrh of the right lung apex.

Liver dullness extends from sixth rib to one inch or more below the costal margin and extends in a curved line across the abdomen, which is otherwise tympanitic. No masses are felt in the abdomen, the skin is wrinkled and pliable, yellowish in hue. Splenic dullness is not obtainable.

June 19, 1898.—Patient's temperature still has the peculiar intermittent character, but he does not seem very ill. Dizziness is better. He appears feeble and has but little appetite; his bowels are regular and there are no bladder symptoms. The urine is normal. He has still a little cough with slight expectoration.

July 6, 1898.—The peculiar temperature continues, being elevated often at night, yet patient does not complain of feeling very ill; sometimes points to head and

speaks of schwindel (dizziness); eats and sleeps well; is down in the yard most of the day.

July 6, 1898.—While patient was in the yard had quite a severe chill. This occurred about 2 P. M. He came into the ward a few minutes later shaking and livid. He now says had similar attacks of short duration before coming to the hospital. He is ordered gr. xx of quin. sulph. daily for three days, followed by gr. x daily for three days. He says that for nearly two years past has lived near the Delaware River, as near as Second Street.

July 8, 1898.—Patient complains bitterly of dizziness. The heart sounds are weak, also the apex beat; in fact the latter is difficult to find, and although we feel that there must be some dilatation of the heart, this is not apparent to percussion. There is evident enlargement of the liver downwards in the mammillary line to one inch below a transverse line drawn on a level with the umbilicus. He complains also now of defective vision, more on the right side.

July 11, 1898.—Patient says that he felt cold all morning.

July 13, 1898.—He still complains of dizziness and coldness, but the temperature has been essentially normal since the 10th inst., falling to 97° at 6 P. M. last evening and at 8 A. M. this morning. We add to the treatment nitroglycerine gr. $\frac{1}{100}$ every two hours, and omit the quinine.

July 16, 1898.—Another rise of temperature occurred to-day, reaching 101°. Patient did not, however, complain of being chilly. Since taking the nitroglycerine pulse is much softer.

July 18, 1898.—There seems sufficient reason to add to our diagnosis "hepatic fever," sustained by the chills and fever, the enlarged liver and yellow coloration of the skin and sclerotics, which we think deepens after one of these spells. We do not feel the gall-bladder. The liver also, so far as it can be palpated, seems smooth. The edge is felt as low as the umbilicus in the median line. An examination of the blood for the malarial organism proves negative. The case lacks, however, the usual paroxysmal pain of hepatic fever, though there is a vague history of abdominal pain.

July 28, 1898.—There have been no chills since July 17th. Since then the temperature has ranged sufficiently near the normal to be regarded as practically normal. He still feels miserable. Complains of general discomfort.

August 1, 1898.—Since July 30th the patient has had a rise of temperature without a chill. His temperature at 7 P. M. on the 30th was 102°. Since then, with exception of some trifling intervening irregularities, it has been normal. He also has some troublesome cough.

August 3, 1898.—Patient is evidently declining. He is dull and apathetic. Very large gurgling râles are heard below the right clavicle; posteriorly are moist râles.

August 5, 1898.—There are still moist râles in the upper part of the right side in front, and evident dullness just below the inner end of the right clavicle. Below the first rib the percussion note is higher pitched on the left side where the inspiratory sound is very harsh but unaccompanied by any moist râles.

August 8, 1898.—Patient has gradually grown weaker since last note, and the jaundiced hue has been growing plainer. Died this A. M.

Autopsy by Dr. Rosenberger.—Body of an extremely emaciated male; decided jaundiced hue over the whole body, more marked on the face, neck, in the eyes, upper extremities and scleræ. There is well-marked barrel-shaped chest.

The muscles of the abdomen are pale and of a greenish hue. The subcutaneous tissue is scant. The intestines are nearly colorless, especially the smaller bowel. There are a few adhesions of the omentum in the right inguinal region near the cæcum, and also near the region of the liver.

The left pleura contains about one-half liter of blood-stained fluid. The right pleura contains numerous adhesions, anteriorily and posteriorily. The pericardium is thickened, covered by a quantity of adipose tissue, and its cavity contains about 90 c.cm. of bile-stained fluid. The heart is slightly enlarged and presents several small milk spots. The right auricle is dilated and contains a large chicken-fat clot which extends into the greater vessels. The aortic valves are thickened, and on the middle leaflet is a small vegetation. The heart muscle is pale and flabby. The aorta is dilated and presents beginning atheroma. The coronary arteries are atheromatous.

The left lung presents numerous adhesions of the outer surface and between the lobes. The apex is solid and grayish white in color; the lower lobe is solid and of a dull red color. The rest of the lung is œdematous, and on section of the bronchial tubes, pus exudes. Right lung at the apex shows areas of croupous pneumonia in the red stage. The remainder of the lung substance is œdematous. No tubercle demonstrable. The spleen, 10 x 7 x 2 cm. Pulp is normal, the capsule thickened, of a dull white color, and on one end presents possibly a supernumerary spleen measuring 4 x 3 cm.

The left kidney (11 x 6 x 2½ cm.) presents an irregular surface showing areas of old infarcts. Cuts with slight resistance. Pyramids are congested. Cortex slightly narrowed. Capsule is moderately adherent and leaves when stripped a granular surface. Right kidney, 12 x 5 x 2½ cm.; surface irregular, shows areas of infarction. Capsule is markedly adherent. Pancreas normal.

Liver, 24 x 19 x 6 cm.; upon section it is found bile stained. Substance is firm, probably beginning cirrhosis. Gall-bladder extends 2 cm. below the margin of the liver; it contains a small amount of bile and one calculus. The common duct is markedly dilated, measuring 2 cm. in width. At the junction of the common duct with the duodenum is a stone measuring 2 x 1 cm., ovidal in shape. Does not occlude the passage of the bile.

The above case is another added to the list of ball valve obstruction of the common duct which has been growing since attention was especially called to it by Osler and Fenger. Another was reported from the service of Dr. Frederick A. Packard in this hospital to the Pathological Society of Philadelphia, May 12, 1898. The case now reported presents the usual symptoms of this affection except pain and it would seem there was not obstruction sufficient to cause this, although it was sufficient to cause dilation of the common duct, another constant symptom of the affection.

HERPES ZOSTER AND ITS RELATION TO INTERNAL INFLAMMATIONS AND DISEASES, ESPECIALLY OF THE SEROUS MEMBRANES.

By Roland G. Curtin, M. D.

In September, 1890, I read a paper in the City of Denver, before the American Climatological Association, entitled, "Is Herpes Zoster a Cause of Pleurisy and Peritonitis?" In that paper the histories of two cases were reported, one associated with pleurisy with effusion, and the other coming on with an attack of localized peritonitis. After reporting these cases I asked the following questions:

1. Was the internal disease a zoster eruption of the serous membrane?
2. Was the inflammation of the internal filaments of the nerves communicated to the pleura and peritoneum?
3. Did the internal inflammation cause the zoster?
4. Were both troubles independent and simply coincident?

I have since that time had the opportunity of observing other cases, which may assist in settling at least two questions, and perhaps the first. I will repeat the two cases given in that paper and then proceed to give other histories collected since which serve to bear upon this subject.

Case I.—*Herpes Zoster with Pleurisy and Effusion.* (Quoted from my former paper.)

Some time ago I had under my care a maiden lady who, in answer to the question, "How old are you?" replied in a low voice? "To you I am sixty, but to other people I am fifty-six." She was in the last stage of locomotor ataxia, so that she could scarcely walk. While in this condition she was attacked with a short, intermittent, neuralgic pain in her side, at the left base of the chest. A careful examination failed to develop any physical signs of pleurisy. In time the pain was followed by a well-marked zoster, which satisfied me that the pain was preliminary to the herpetic eruption. About the time this eruption appeared I noticed a pain with every inspiration, like the "catching pain" of pleurisy, associated with considerable constitutional disturbance, and a dry, hacking cough. A little later my attention was called to the diminished movement of the left side of the chest, which was almost immobile, and a careful physical examination

revealed an effusion which half filled the side of the chest. This effusion continued until the chest was entirely filled. In this case the zoster and the plurisy seemed to be coincident.

CASE II.—*Herpes Zoster with Dry Pleurisy.* (Reported by Dr. H. H. Doan, Resident Physician of the Philadelphia Hospital.)

Michael Dougherty, aged 42; white, painter, single; weight, 120 pounds. Family history revealed no facts of any interest.

Previous History (November 29, 1891).—Used whiskey to excess. Had initial lesion, which from history given was evidently specific; malarial fever seven years ago; erysipelas twice; inflammatory rheumatism; friction rub heard on right side of chest five years ago; has been a patient in the drunkards' ward three times for delirium tremens.

Two weeks ago he had a severe catching pain in the right side; it felt like a stitch. He never went to bed. Five days later a number of lesions appeared along the track of the seventh or eighth intercostal nerve of the right side, evidently an attack of "shingles" following pleurisy. Temperature was taken for one week. It reached 99.30 once. In this attack, which was an evident case of pleurisy at the seat of a former pleurisy, which he had five years before, it would be difficult to tell positively whether the pain of four weeks ago was from the affected nerves or the inflamed pleura.

CASE III.—*Zoster and Pleurisy.*

Prof. George H. Rohe, of Baltimore, having had his attention drawn to the subject under consideration by my first paper, in 1890, informed me that he had in his hospital a case of herpes zoster of the chest with a marked pleuritic friction sound underneath the seat of the eruption. The friction sound was so well marked that he used the case to demonstrate the sounds of pleurisy to his students.

Dr. Rohe could not tell which was the initiatory affection in this case.

CASE IV.—*Herpes Zoster and Pleurisy with Chronic Bright's Disease.* (Reported by Dr. P. Janney, Resident Physician, Philadelphia Hospital.)

Bridget McC., white, female, aged sixty years, born in Ireland, housewife, married.

Family History.—Father and mother died of old age. One brother died at the age of seventeen of "enlargement of the liver." Four sisters and one brother living and healthy.

Previous History.—Healthy as a child. In 1845 had influenza with the rest of her family. In 1848 had typhus fever and was sick three or four months. In 1856 had jaundice for three months. No history of biliary colic. Has had one living child and three miscarriages. No venereal history. Five years ago her feet became swollen, had pains in back, passed frequently small quantities of urine. Ever since has had more or less swelling of feet. Admitted to the Philadelphia Hospital about two years ago with diagnosis of Bright's disease.

History of Present Disease—In the middle of October, 1891, she felt a catch at intervals in the right side in the nipple line. October 30, 1891, had a constant severe pain in the seventh and eighth interspaces on the right side, and a friction rub was heard on November 2d. Chest was examined and a harpetic eruption was found in the seventh and eighth interspaces, extending around almost to the sternum and over the seventh and eighth dorsal vertebræ. There was a constant pain and a slight moist crackle heard on inspiration and expiration.

Examination of Urine.—Urine passed in large quantities; pale and of low specific gravity, containing a slight trace of albumin, but no casts; acid in reaction. All physical signs disappeared in two weeks, showing that the attack of pleurisy was acute. The pleurisy in this case seemingly preceded the external eruption.

CASE. V.—*Herpes Zoster Complicating Phthisis and Chronic Bright's Disease.* (Reported by H. H. Doan, Resident Physician, Philadelphia Hospital.)

John Hogan. Diagnosis, phthisis and Bright's disease. A native of Ireland, tailor by occupation, single, tall and thin.

Family History.—Mother asthmatic for many years. Father died of Bright's disease. No tuberculous history.

Health, usually good; drank very heavily and used tobacco to excess. No venereal history. Small-pox in 1866.

Had cough for two years, losing flesh; expectoration very slight until May, 1891. In January, 1891, had a continued attack of diarrhœa. July 20th, noticed eruption on left side of the upper part of the abdomen; no pain. Before this, although following the appearance of the eruption, there was severe pain, lasting two weeks. At one time during this attack he had slight hæmoptysis, lasting for one day. Lost apetite; bowels constipated. On admission to hospital there was an herpetic eruption extending from spine at junction of fourth rib around to the nipple, with points of tenderness. No friction sounds heard, nor could fluid be demonstrated.

Since admission he has had frequent chills, occurring at irregular intervals, with decided irregularities of temperature. No plasmodium found in blood.

Urine, acid and albuminous, with many granular and hyaline casts. Numerous bacilli found in sputum.

With tubercular phthisis we have more or less plastic pleurisy, and many observers have reported cases of zoster complicating tubercular disease of the lungs, and in their cases the pleurisy antedated the eruption.

CASE VI.—*Herpes Zoster with Localized Peritonitis.* (Quoted from my former paper.)

I had a case which served to bear upon the subject under discussion—apparently an inflammation occurring in another serous membrane simultaneously with zoster.

A women, thirty-five years of age, who was about four months pregnant, had what I had diagnosticated as a localized peritonitis, occurring on the right side of the upper part of the abdomen, just below the margin of the liver. There appeared later at the same position a well-marked, clustered eruption. At about the same time a deep-seated soreness and tenderness appeared, extending to the median line.

The febrile symptoms were marked; respiration and movement caused a great increase in the pain, which was constant; and pain was caused by the flatus, which was present, passing through the bowels. She barely escaped aborting. Later on the uterus increased in size and caused much pain, which was relieved, but not entirely so, when she was delivered. It was some months before the soreness entirely disappeared. The diseases seemed to be coincident in this patient.

CASE VII.—*Herpes Zoster following Catarrhal Appendicitis.*

Mr. L. had a sharp attack of catarrhal appendicitis with high fever, which ran its course in about seven days. The next week a sharp, lancinating pain appeared, followed later by a zoster eruption on the right side of the abdomen, the right hip, and the upper part of the thigh. The localized peritonitis preceded the skin affection in this case.

CASE VIII.—*Herpes Zoster following Arthritis and Effusion of Right Knee.*

Judge A. Right knee-joint was very swollen and painful from rheumatic arthritis. This condition had continued for several weeks when an eruption of herpes zoster came on the outside of the knee and popliteal space, and ran the usual course of that affection. The eruption did not encircle the knee.

Here we find a case of hydro-arthritis of the knee-joint of six weeks' standing, the skin around it being secondarily affected by zoster.

CASE IX.—*Influenza; Meningitis; Pleuro-Pneumonia followed by Herpes Zoster.*

F. L., had an attack of influenza followed by meningitis and later on by a pleuro-pneumonia of the right lung. Late in convalesence he had a marked attack of herpes zoster situated on the right side of the lower anterior chest and upper part of the abdomen.

CASE X.—*Hematuria and Zoster.*

S. A., had in May, 1899, an eruption of well-marked zoster over and above the position of the left kidney, extending around to the umbilicus and below it. In the third week of August, two and a half months later, he began to pass blood in his urine. This continued for months. There was no pain or other discomfort over the kidney or bladder, except when he passed a clot, and then he felt a slight pain and discomfort at the symphysis pubis.

The quantity of blood constantly varied and continued for months. The color of the blood was generally dark or quite black; he had no constitutional disturbances. Two years ago, September, 1897, he had a similar eruption on his face, which lasted about ten days or two weeks; at the same time he told the doctor that his urine was high colored. The doctor examined it and said it was blood. This hemorrhage lasted four or five weeks; he never had any eruption or hematuria at any time other than these two occasions.

RECAPITULATION.

1. In Cases, 1, 2, 3 and 6, the herpes zoster seemed to come on at the same time as the internal inflammation.

2. In Cases 4, 7, 8 and 9, the affection of the serous membrane preceded the eruption.

3. In Case 5, the zoster appeared during the course of a chronic pleurisy of tubercular phthisis.

4. This résumé seems to indicate that inflammations of the serous membranes precede the zoster; therefore, we may reason that the inflammation caused the eruption.

5. These conditions occur together or follow one another too frequently to be simply coincident.

I feel sure that the cases here reported settle at least some of the problems that I suggested in my former paper:

1. Was the internal disease a zoster eruption of the serous membrane?

2. Was the inflammation of the internal filaments of the nerve communicated to the pleural peritoneum?

3. Did the internal inflammation cause the zoster?

4. Were both troubles independent and simply coincident?

While on this subject of zoster and internal diseases, I will take the opportunity to state that I had a case, in 1874, of œsophoreal cancer that had, late in the disease and over its seat, a complicating zoster eruption encircling the left side of the chest at the margin of the ribs. The eruption, it seemed to me, might be caused by the cancer, as the œsophagus is located to the left of the border of the vertebræ, near the roots of the nerves.

In 1890 to 1893 I had quite a number of cases or influenza and catarrhal fever complicated with zoster, generally coming on with the less acute influenzal attacks. A number of my friends reported to me one or more attacks of the same kind in their cases of acute and subacute influenza.

The subject being, I believe, a new one, I shall feel repaid if the attention of future observers is called to this interesting condition.

PHTHISIS FOLLOWING TRAUMATISM.

By ROLAND G. CURTIN, M.D.

A. Phthisis Following Blows on the Chest.

The six cases that I report in this paper have interested me, and although somewhat dissimilar, I have grouped them together. The first three were the result of bicycle injuries. In all of them the symptoms were preceded by a fall from a wheel, the patient being violently thrown to the ground, striking on the anterior chest, and being found to have no evidence of external injury.

Case I.—Mr. S., aged twenty years, was riding on a country road after dark. His wheel caught in a rut, and he was thrown violently to the ground, striking his right chest anteriorly at the fourth rib near the nipple. He expectorated blood at the time of the accident, and later had a number of profuse hemorrhages, some fever, and evidences of consolidation of the base of the right lung. He is now floating around the world endeavoring to maintain an existence, having chronic pulmonary tuberculosis.

Case II.—Mr. B., aged twenty-one years, was riding his wheel at night. He was thrown as in Case I, striking the anterior chest, left side near nipple. No marks were found on the surface. He had pain of pleuritic character, followed by pneumonia of the left side, and later on by a pulmonary abscess of the same side. He has now evidently latent pulmonary disease.

Case III.—Mr. K., aged twenty-eight years, was speeding down a steep hill towards a railroad crossing. The man at the gate shut it as he appoached. In order to avoid crashing into the gate or the train, he ran his wheel into a bank, and was thrown off. He struck violently on the middle of his left anterior chest. There was no external discoloration. Following this he had pleurisy, and later pulmonary tuberculosis, from which he died fifteen months later.

Dr. Newton,[1] of Montclair, New Jersey, has reported a case of rupture of the heart, following a not very serious blow upon the chest wall in a bicycle collision. That case showed that severe internal injuries may result without the chest wall's showing any evidence of injury. In those cases to which I have called your attention, the pluera, or even the lung, might have been severely injured, giving rise to the symptoms found in them at a later period. Hard pressure and

[1] Report of American Climatological Association, 1899.

marked shocks on the chest do not always, however, cause pleurisy or inflammation of the lungs; this is shown by the infrequency with which foot-ball players suffer from those affections, and yet they are subjected to great pressure and to more or less severe shocks, especially of the chest. On the other hand, it is said that the hard blows on the chest received by pugilists frequently cause tuberculosis. In the injuries received by the patients whose cases are herein reported the body was suddenly thrown to the ground while moving forward with a greater or less degree of rapidity, thus giving a velocity to the body in its impact against the earth. Such an injury would be more likely to cause a contusion of the internal organs than would the shocks received by foot-ball players. I feel quite sure that in the cases here reported the pluera at least was injured, and in one case the lung itself seemed to be also, because free pulmonary hemorrhages set in immediately after the accident.

In analyzing the records of the prize-ring, it was noticed by the late Dr. W. L. Axford that traumatism plays a part in the production of the phthisis, which is notoriously common among prize-fighters. Lebert and Scholtz had published clinical observations which tended to show that traumatism may become the direct cause of pulmonary tuberculosis. Mendelsohn, Bremer, Jaccoud and others, have pointed out that their observations confirm those of Lebert and Scholtz. Dr. Eloy stated, "that a person previously in good health, who sustained a traumatism of the chest wall without an external wound, soon afterwards developed symptoms of pneumonia and hemoptysis, which later assumed the character of acute phthisis. It is possible that in some cases the injury merely disturbs the circulation and starts inflammation, which favors an attack of tuberculosis." Mendelsohn suggests that the tuberculosis may be engrafted into the patient in the hospital to which he is taken for treatment. This would not apply to my cases, for they were all treated at home. In Dr. Eloy's opinion, "The most acceptable view, in our present state of knowledge, is that the injury produces a modification of the nutrition of the pulmonary tissues, which thus affords a more favorable nidus for the growth of the bacilli. The occurence of phthisis after truamatism of the chest, without external wound, must be regarded as an established fact, and hence appropriate prophylactic measures are called for in all cases of chest injury, especially if there is a family history of tuberculosis."

In one of my cases, tuberculosis followed the injury to the lung; in the second, the injury was probably only of the pleura; the third showed evidence of disease of the pleura and lung some time after the accident in a pleuro-pneumonia, and later by a pulmonary abscess; finally it resulted in a chronic ulceration of the lung.

The fact should not be lost sight of that pugilists are particularly tempted by sexual and alcoholic excesses, with loss of rest, which may be the cause of much of the tubercular disease found among them. Moreover, the evidence of lung disease does not usually date immediately from the time of the fight, but later on when in the midst of their exercises.

B. Phthisis Following Abscesses of the Hands, which Resulted from Traumatism.

I have seen two cases of infected wounds of the hand—one of the right and one of the left—which were followed by abscess formation and imflammation of the lymphatics of the arms and axillæ. Later on there were symptoms of abscess of the lung, corresponding to the side in which the infection originally took place in the hand.

In one case evacuation promptly took place through a bronchial tube, and in the other, which occured in a man with tubercular history, the abscess was soon followed by tubercular disease, which in time involved both lungs and caused death.

C. Phthisis Following a Foreign Body in the Bronchial Tube.

In the sixth case a bone was swallowed, which passed into a bronchial tube. An irritative, hoarse cough followed immediately after the swallowing of the bone. Two months later the patient began to spit purulent matter, and, at times, altered and streaked blood. Four months after swallowing the bone she coughed it up. It measured half an inch in length and was pointed at one end; the other end measured one-fourth of an inch by one one-half inch. A little over a year later she was seen in a dying condition, with copious expectoration, containing numerous cocci of suppuration, pus and lung tissue.

ULCERATION OF THE TRICUSPID VALVE.

By JOHN H. MUSSER, M.D.

The following case is worthy of record because of its rarity. It is unusual to see such extensive right-side endocarditis and valvulitis. The sequence of events seems to be endarteritis, attrition of roughened surface, ulceration, possibly secondary infection of the bruised and abraided surface. It is to be regretted no bacteriological studies were made, as it also looks like trauma and inflammation without infection, as indicated in Virchow's recent communication. The lesion was not diagnosed during life.

J. B. P., aged seventy years, male, native of Pennsylvania, resident of Philadelphia, widower, laborer.

No tuberculosis, alcoholism or rheumatism in family history. Patient was a moderate drinker, according to his own statement. Denied specific history. Had had typhoid fever in youth. Never rheumatism. Always been a healthy man. Worked hard at manual labor all his life. Admitted to Philadelphia Hospital, medical ward, May 9, 1896. Was a tall man, somewhat wasted. Hair scant and gray. Arcus senilis marked. Great atheroma of temporals and radials. Mental condition somewhat childish and statements probably not much to be depended upon. Was without friends, and history only what patient gave upon admission.

Some slight œdema of lower extremities, but this was never marked. There was an ulcer upon the third toe of right foot, and that member was blackish, with sensation lost and appeared gangrenous. Heart dullness; right border extended as far to the right as the right mid-clavicular line and was evidently due to a distented right auricle. Left border to left mid-clavicular line. There was a loud systolic murmur in aortic area transmitted upward to vessels of neck and downward to apex. No murmurs heard in tricuspid area. Heart's action was slow and somewhat irregular, and the muscular element in the first sound was almost lost. Pulse, 70-60.

Lungs.—Expansion poor over both apices. At the bases posteriorally the breath-sounds are harsh and broncho-vesicular in character. A few dry râles are heard over the upper lobes.

Urine.—Amount varied from time of admission to death from 25 ounces to 30 ounces in the twenty-four hours. It showed a trace of albumin, and the specific gravity ranged about 1020. Temperature on admission was 96°. It ranged from 99° to 96° up to May 21st, when it sunk to 93.4°, then rose abruptly to 102.2° May 22d and then sunk to 100° May 25th, the day of his death. The patient showed symptoms of uræmia, stupor, headache, and muttering delirium, gradually failing till death.

Autopsy, Twenty Hours after Death.—The body of a large, emaciated man. Rigor mortis well marked. Some cadaveric lividity. Gangrene of the third toe of the right foot, where there is also a trophic ulcer. The heart is much enlarged and dilated by clots, which are especially marked upon the right side. The pulmonary artery contains a long thrombosis. Weight of heart, five hundred grammes.

The coronary arteries are both diseased and dilated and tortuous. The anterior coronary is one centimeter in diameter at its commencement. Both arteries show advanced sclerotic changes and beginning calcareous infiltration. The muscle of the left ventricle measures 25 mm. in thickness. That of the right ventricle 6 mm. The heart muscle is pale and friable and shows a considerable increase in its connective tissue elements with several small scar-like areas. It also shows marked fatty degeneration. There is a calcareous nodule measuring 10 x 8 mm. in the wall of the left ventricle in the auriculo-ventricular ring behind the insertion of the posterior mitral leaflet. The aorta is exceedingly atheromatous down to the insertion of the aortic valves, and the valves themselves are studded with calcareous nodules in both the inner and outer surface. There is some adherence of two of the leaflets, but besides this very little evidence of an acute inflammatory process or impairment of function. The mitral valve is somewhat thickened and sclerotic along the free border, but not as extensively as the tricuspid, and the orifice is not narrowed, nor does it appear to be incompetent. Pulmonary valves normal. Tricuspid valves show a marked fibrous change along the free edge. The posterior leaflet is particularly affected, and the border is studded with several large and hard fibrous nodules. On the ventricular surface of this leaflet is an ulcer 10 mm. by 5 mm. The edge and floor of this ulcer are ragged, and it appears to have been formed by erosions caused by the rubbing of the valves against the ventricular wall. There is no evidence of an acute inflammatory process at any time.

Lungs.—Very considerable hypostatic congestion and œdema of both lower lobes posteriorly. Some old scars containing chalky nodules at the apex of the left lung.

Kidneys.—Both are cyanotic and sclerotic.

Liver.—Cynotic and sclerotic.

There are very extensive atheromatous changes in all the arteries of the body, especially affecting the splenic, which is externally tortuous and elongated.

Microscopic Examination.—The portion of the valve leaflets nearest the base of the valve is greatly thickened by an increase in its connective tissue elements that affect particularly the portion of the leaflet bordering upon the ventricular surface. An ulcer occupies the more prominent portion of the ventricular surface of the swelling. The edges are sloping and the floor ragged. There is an area of tissue bordering upon the ulcer and forming the floor and extending some distance into the dense connective tissue about it that shows a very considerable amount of fatty degeneration. The immediate floor is formed by frayed and fibrous tissue and is covered by fatty detritus.

There are several collections of calcareous granules in the tissue bordering upon the ulceration. The endocardium of the auricular surface about the point of contact with the other leaflets shows some round-celled infiltration and suggests a recent inflammatory process.

TWO CASES OF UNUSUAL FORMS OF LEAD POISONING.

By F. X. DERCUM, M.D.

Lead poisoning is an affection so exceedingly common that cases presenting variations from the typical symptoms are always interesting. The first of the cases here reported is peculiar in that the poisoning occurred in an epileptic and that tremor was present as a striking feature. Tremor is, of course, known to occur in lead poisoning, but it is infrequent. In this case it formed the most prominent symptom. The history is briefly as follows:

CASE I.—J. J. A., white, aged forty-six years; married; occupation, carriage painter. Admitted to the the nervous wards of the Philadelphia Hospital, September, 1898. The family history is unimportant. Father died of pneumonia, aged forty-four years; mother died of erysipelas, aged seventy-seven years. One brother died of pneumonia, aged twenty-one years; another brother living and well. Two sisters died in childhood, cause unknown.

Personal History.—Had no other disease of childhood save chicken-pox. At the age of six or seven years he became subject to peculiar attacks. States that the attacks would come on with a rumbling in the ears, and that following this he would at once start to run. Two balls of fire would appear before his eyes, and, after crying and being petted awhile, the attack would pass off. During the attack he did not know what was going on around him. All that he could remember afterward was the two balls of fire. He never fell during such an attack and never had a convulsion. These attacks occurred at intervals until he was twelve years of age. From this time on until he was twenty-one he was absolutely free from attacks of any kind. At the end of this period, however, attacks of a different nature came on. Instead of running, as he had in childhood, he would suddenly wander off, and, after a period of several hours, would suddenly become conscious and realize that he was in a strange place. After one of these attacks he would have no recollection whatever of what had occurred. Between twenty-one and thirty years of age, these attacks would recur every four or five weeks. They sometimes lasted as long as twenty-four hours, though they never exceeded this time. Subsequently, the attacks occurred at much longer intervals, and of late years they have only been occurring about every six months. About one and a half years ago, one of these attacks lasted three days, and when he regained his normal mental state, found himself in Doylestown, without any knowledge as to how or why he had come there. He states that there was no reason why he should have selected Doylestown, for he was not acquainted there nor had he had any business transaction there. He says that of late years the tendency has been for the duration of these peculiar seizures to increase.

At twelve years of age had typhoid fever and claims to have passed through a second attack at the age of sixteen. Subsequent to this, he had a weak and irritable stomach, but otherwise was healthy with the exception of the peculiar running seizures, mentioned above.

Eight years ago he suffered from lead colic. From this he appeared to make a good recovery. Six months later he had a second attack of lead poisoning and this time his arms became paralyzed, the right being more affected than the left. Wrist drop existed in both arms, and, in addition, he could not extend the right arm at the shoulder. He could partially extend the left arm. This condition persisted for about two years. At the end of this time he was able to resume his work as a carriage painter. No new symptoms made their appearance until five months ago. He then began to have trouble with his right arm and right leg. He awoke one morning with marked numbness extending throughout the entire right arm and right leg. Considerable weakness also made its appearance in these limbs. Both the numbness and the weakness persistently increased in severity, but he was able to continue at his work until two weeks ago.

For the past three years he has been resting poorly at night. After he goes to bed he begins to worry about the events of the day and feels as though he has left undone the most important things. Says he has dreams every night, but his dreams present no particular character. At times, when awake at night and in the dark, he has visual hallucinations; says that he frequently sees cats and dogs running before him; adds, that while he sees these animals clearly, he fully realizes that there are no such creatures present. In reply to questions, he states that he has never had an auditory hallucination except the rumbling preceding the attacks in childhood.

Upon admission he complained especially of loss of power in the right leg and right arm, and to a less extent of loss of power in the left arm. A tremor was present, involving both hands and arms, and which appeared to be constant. There was occasional tremor of the head and lips. Pain was complained of in the muscles of the arms and in the knees.

Examined, September 20, 1898.—The patient is well developed. When standing with toes and heels together and eyes closed, his sway is slightly exaggerated. His gait is somewhat uncertain. If he attempts to turn suddenly while walking, he stops, becomes unsteady, but does not fall. No paresis is present in either leg. The patellar jerk is present in both sides and is decidedly increased; it is more marked on the left side. No ankle or patellar clonus is present on either side. The plantar and cremasteric reflexes are present on both sides. No biceps-jerk. No jaw-jerk.

The tongue is tremulous, clean, tooth-marked and extended in the median line. The pulse is regular. There is increased tension and some atheroma of the vessels.

Both legs can be flexed and extended at the thighs. Both thighs can be rotated. Both knees can be flexed and extended, as can also both ankles. The toes can be flexed at the metatarso-phalangeal joints, but at none of the phalangeal joints. The legs can be abducted and adducted; they can be elevated and held fairly steady. When the movement begins, however, there is a tremor of the thigh muscles, slightly more marked in the left limb. There is no demonstrable ataxia. There is no apparent wasting of lower extremities.

The forearms can be flexed and extended on the arms. The hands can be flexed and extended on the wrist, but there is some weakness of the extensors. The lateral movements of the wrists are normal. The arms can be raised over head and all other ordinary movements executed. The fingers can be flexed, but if the effort be repeated one or more times they become affected with spasm and become fixed in position as in a contracture. At first, this spasm can be overcome by an

effort of the will, but at other times force is required to "unlock" the hands. The index finger of the left hand is somewhat more readily affected by this spasm than the other digits. The patient states that at times his fingers become locked, necessitating manipulation of both hands to separate them. In both of the upper extremities there appears to be more or less wasting. The muscles of both upper arms appear wasted. This is true of the biceps, triceps and deltoid. There is some flattening of the muscles of the inner and upper portion of the forearm. There is also wasting of the muscles of both hands, especially of the hypothenar eminence, most marked on the right side. The least change is seen in the forearms. The grip of the left hand is fairly good, that of the right is weak. No wrist drop is present at the time of the examination.

Tremor is noticed, more particularly in the hands. When the latter are relaxed or at rest, this tremor is hardly apparent, but upon voluntary motion or if the patient's attention is attracted to the hands, the tremor becomes marked. It is most noticeable when the hands and arms are extended. It is a regular tremor, small in extent and of about the same degree as that noticed in paralysis agitans. Tremor of the lips is not noted at present, but it was quite evident at the time of admission. The patient calls attention to the fact that his tremor is not constant in degree, and that it varies considerably with excitement. He further states that tremor was first present for about a year, and that it subsequently disappeared, but recurred again two weeks ago. It was simultaneously in both upper extremities and, in addition, affected the lips, head and right leg.

The patient complains of pain in his arms, which he describes as "sore pain." Complains also of pain in his knees, which he describes as "rheumatic." These pains came on about the same time that he lost power in his arms. They persisted for several months, then disappeared but recurred again recently. At the time of the examination they are only present in the morning, and toward noon pass away.

Cutaneous sensibility is preserved in all forms over all parts of the body. The patient distinguishes clearly and readily between hot and cold, readily appreciates painful impressions and accurately distinguishes between sharp and blunt points.

The heart and lungs are negative. There is some tenderness over the epigastrium and there are other evidences of chronic gastritis. There are no blue lines on the gums, though a marked blue line, the patient states, has been present upon former occasions. An examination of the urine reveals the presence of a small quantity of lead.

Examination of the eyes by Dr. Charles A. Oliver, reveal the following conditions: O. D., one-third vision; O. S., one-third vision. Pupils are equal, both react slightly to light. Reaction to accommodation and convergence normal. Upon lateral rotation, there are ataxic movements of both eyes at both canthi, slightly more marked upon rotation toward the right. On vertical rotation, ataxic movements are present at both upper and lower limbs. There is slight tremulousness of the orbicularis, when the patient brings the lids close together. The retinal arteries are slightly reduced in both eyes. The eye-grounds are otherwise normal.

The above case is extremely interesting, both on account of the seizures from which the patient suffers and on account of the peculiarities presented by the symptoms of lead intoxication. The attacks described as occurring in childhood were evidently those of running epilepsy. Those occurring after twenty-one years of age suggest double consciousness, for during these attacks the patient for hours and even days performed automatic acts as are ordinarily performed in the normal

mental state. The duration of the periods of abnormal consciousness was always several hours, and upon one occasion extended over three days.

We have evidently before us a case in which lead poisoning occurred in an individual already neuropathic, and in whom the symptoms of the poisoning assumed the rather unusual form of tremor and of spasm of the flexor muscles of the fingers. The symptoms of weakness described by the patient as affecting especially the right arm and leg were confirmed only in the right arm. In the right leg this symptom appears to have been merely subjective. This appeared also to be true of the numbness of the right arm and leg, as tactile and thermal sensations were intact.

The second case is interesting because of the unusual distribution of the palsy.

CASE II.—The second case is that of J. P. W., aged forty-five years; married; laborer in lead works. Was admitted to the nervous wards of the Philadelphia Hospital, September, 1897. At that time complained especially of paralysis of the shoulders.

The family history is unimportant. Father and mother died of unknown causes. One brother and sister died in infancy. One brother and three sisters are living and well.

Personal History.—The patient had measles and whooping-cough in childhood. When thirteen had typhoid fever. Denies having had any venereal disease.

History of the Present Illness.—He had been working for the past eight years in a paint factory. Seven weeks ago he began to have a feeling of weakness in his shoulders. Also suffered at this time from cramp in his stomach and constipation. The pain in his shoulder steadily increased, and five weeks ago he became utterly unable to raise the arms at the shoulders. From the time that the weakness was first noticed up to two weeks before admission, he felt darting pains in both shoulders, especially when he attempted to move the arms. He could always move his forearms well in all directions, and also his wrists and hands.

Examination, December 12, 1897.—Marked general weakness was noted. The muscles of the neck and both rhomboids and trapezii are intact, but the deltoids are markedly atrophied. The patient is utterly unable to extend arms at the shoulders. In addition, both the triceps and biceps muscles are wasted, though both extension and flexion of the arms can be performed, but these movements are weak. The forearms appear small, though no wasting is present. However, both the flexor and extensor groups of muscles are weak, and this weakness is slightly more marked in the extensors. There is no trace of wrist drop and there is no wasting of the intrinsic muscles of the hand. The tendon reflexes of the arms are absent. The knee-jerks are both plus. There is no ataxia of the arms and legs. Both legs are markedly weak. The patient is unable to stand for any length of time, and can only walk with difficulty. Sensation is everywhere intact. There are no pupillary anomalies. The eye-grounds were not examined. The gums reveal a blue line.

In this case the marked atrophy of the deltoids, triceps and biceps muscles, together with the preservation of the extensors and other muscles of the forearms and hands and the absence of wrist-drop, is the interesting feature. Involvement of the deltoids and other arm muscles is, of course, known to occur in lead poisoning, but it is unusual, especially with absent wrist-drop.

NOTE ON THE RELATION OF THE "SPACING SENSE" TO ASTEREOGNOSIS.

By F. X. DERCUM, M.D.

As was long ago demonstrated, by Hoffman, a large number of factors enter into stereognostic perception. The writer has separated these for practical purposes into the tactile sense, the temperature sense, the pain sense, the pressure sense, the sense of weight or muscular sense, the sense of location (that is, the ability of the patient to correctly refer an impression to the area touched), the knowledge of the position of the fingers, the condition of the fingers and hands as regards mobility, and lastly, but most important of all, the ability of the patient to distinguish one or more impressions made upon the hand or fingers at one and the same time. To this faculty the writer has given the name of the "spacing sense." It deserves a special designation, because the tactile sense may be well preserved when the "spacing sense" is lost. The function implied by the "spacing sense" is doubtless cortical.

The "spacing sense" was studied in forty-one cases of hemiplegia; it was found preserved in twenty-one, and in these twenty-one cases stereognostic preception was also preserved. In one case in which astereognosis existed the "spacing sense" could not be studied because of the co-existence of aphasia. In seven cases it was found decidedly diminished, the patient either being unable to distinguish two impressions, save at unusual distance or making occasional errors. In these seven cases partial astereognosis existed. In ten cases the "spacing sense" was lost, and in these ten there was also complete astereognosis. In one case the "spacing sense" was merely diminished, and, notwithstanding, there was complete astereognosis. In the remaining one case the "spacing sense" was fully preserved, but, notwithstanding, the stereognostic sense was lost; in this case, there was, however, a decided loss of the ability of the patient to correctly describe the position of the fingers when the eyes were bandaged, a decided loss in the power

to correctly refer the cutaneous impressions to the parts touched, and a marked secondary contracture of the hands and fingers. In addition to the fully preserved "spacing sense" there was, in the case, also, a fully preserved tactile sense, a slightly diminished temperature sense, a preserved pressure sense and a very good power to estimate weight. These facts prove, of course, that the "spacing sense," while exceedingly important to the preservation of stereognosis, does not prevent stereognostic loss when other faculties are impaired. It is exceedingly probable that in the case in which it was fully preserved, together with stereognostic loss, the inability of the patient to determine the position of the fingers was a factor of prime importance in causing the astereognosis. This is also evidenced by three cases of locomotor ataxia in which complete stereognostic loss existed; the "spacing sense" was lost in only one, and merely diminished in two. In these ataxic cases the ability to recognize the position of the fingers was lost or greatly diminished in all three. Here, evidently, as in the single case of hemiplegia above mentioned, the loss of the knowledge of the position of the fingers appears to have played a rôle, as important, if not more important than the loss of the "spacing sense."

In two cases of ataxic paraplegia in which stereognostic loss existed the "spacing sense" was lost in both, and this was also true in a case of multiple neuritis similarly affected. In the case of multiple neuritis, however, the loss of the "spacing sense" was accompanied by a decided tactile hypæsthesia, and was doubtless in this instance peripheral in origin.

The above results indicate unmistakably the importance of the "spacing sense" to the preservation of the stereognostic sense, though, as has been stated, in rare instances it may be preserved when the stereognostic sense is lost. In such cases, the astereognosis appears to be due to the loss of the knowledge of the position of the fingers, ataxia of movement and inability to properly refer the impression to the part touched, though on the whole these factors, as well as the ability to perceive mere tactile impression and the pressure, temperature and pain senses, appear to be relatively less important.

NOTE ON HEMIPLEGIA WITH ŒDEMA OF THE PARALYZED ARM.

By F. X. DERCUM, M.D.

Thomas McN., white, aged sixty-six, married; birth place, Ireland; occupation, laborer; admitted November 6, 1898.

Family History.—Father and mother died at advanced age. No nervous diseases in the family. Has five living children, all healthy. One son died of phthisis.

Past History.—Many years ago he had an attack of erysipelas, and later on "bilious fever." Admits alcoholic excesses. No venereal diseases.

Present Illness.—About fifteen weeks preceding admission the patient, while walking, suddenly fell to the ground, but did not lose consciousness; immediately, it was noted that he had lost power in left extremities. The face, he claims, was not involved. The left hand subsequently became swollen, but patient cannot remember when.

Physical Examination.—Patient is a man of rather large physique, complexion florid, bones large and prominent. Considerable mental impairment is manifest, and patient's statements cannot be relied on.

The pupils are equal, medium size, irides react normally, extra-ocular movements seemingly normal. Apparently no hemianopia. Tongue broad and flabby, protruded with slight deviation to the left. Pulse is weak, though regular, the radial artery is markedly hard and rough. No evidence exists of facial paralysis. The speech is thick, but there is no aphasia. The chest is emphysematous in character; there is great symmetrical prominence, and tympany exists almost universally over the anterior surface of the chest. Scarcely any movement of the chest walls is visible on respiration. Respiratory sounds feeble. The heart sounds are feeble. The first sound is replaced by a loud blowing murmur, heard over the apex, but more distinctly over the vessels of the neck. The abdomen is decidedly scaphoid, largely due to the prominence of the chest wall.

The power of movement is intact in the right extremities; in the left it is nearly lost. There is absolutely no power of movement in the left arm, but the leg can be slightly flexed with much effort at the knee. It cannot, however, be raised from the bed, abducted or adducted.

There is no anæsthesia. The knee-jerks are both plus. There is no biceps or triceps jerk. Right plantar reflex marked, left absent.

A notable feature of the case is that of a very marked œdema of the left upper extremity. The œdema is especially great in the hand, especially the back of the hand and fingers, the latter being much enlarged. The œdema is marked throughout the entire forearm, and

extends into the upper arm up to the junction of the middle with the upper third of the arm. Great pitting is noted on pressure. There is no œdema elsewhere, and above the area of swelling the arm is perfectly normal. The area of œdema and the normal area above it are quite sharply delimited.

Localized swelling or œdema of the extremities in hemiplegia is excessively rare, and thus far is without explanation. A similar case has been placed on record by Dr. H. A. Hare, *Journal of Nervous and Mental Diseases*, 1899.

TYPES OF CEREBRO-SPINAL SYPHILIS, SHOWING CHARACTERISTIC MULTIPLE LESIONS, AND ESPECIALLY INVOLVEMENT OF CRANIAL NERVES.[1]

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The following cases are reported in order to illustrate some of the multiform phases of cerebro-spinal syphilis. They illustrate very well the fact that this disease is not confined to any one part of the nervous system.

Lewis Finnegan, white, aged thirty years; birth-place, New York; residence, Philadelphia; occupation, machinist; admitted June 25, 1898.

Chief Complaint.—Paralysis of both legs, retention of urine, incontinence of fæces and failing vision in right eye.

Family History.—Father died at thirty-eight years of age, of liver trouble; mother living and well at sixty-one; one brother died of diphtheria. One brother and sister living; brother has stomach trouble. Sister has been in Norristown six months (insane), cause supposed to be parturition. Had one uncle who had temporary insanity.

Social History.—Married, has one child three years old. Mother and child healthy. Eight years ago used alcohol to excess for two years. Has used tobacco since a boy.

Past Medical History.—Had ordinary diseases of childhood; eight years ago had a chancre, which was followed by a bubo, mucous patches and a papular eruption on chest. Has suffered with nasal catarrh for years. Seven years ago had nose "burned out," and since then has lost his sense of smell.

Present History.—First noticed one year ago numbness and pain in right side of face with failing vision of same eye. When trouble with eyes began, patient went to Wills' Eye Hospital once a week. On one occasion he was told the eye must be removed at once. He was to return to the hospital that evening, but, owing to an accident, did not. Eye improved after that. He next had pain in right side of cranium; this lasted for three months. Pain then ceased, and now has areas of anæsthesia on right side of face.

Prior to the eye symptoms, which began a little over one year ago, he suffered intensely with neuralgia on the right side of face. The pain was attributed to the teeth, and he was advised to have them all extracted.

[1] The clinical reports in this paper are entirely the work of Dr. Landis, and he is entitled to full credit for them.—J. H. L.

About eleven weeks ago awoke at night and had a paralysis of both arms and larynx. This condition lasted nearly two weeks, when recovery of power in arms took place, and great improvement in the larynx. Soon after this (two or three days) he noticed a weakness of left leg, which has steadily increased until motor power is lost. Then noticed prickling and numbness in right leg, with increasing weakness. Bladder and rectum became incontinent four days ago. Has had severe pain between the shoulders since last March. This was followed by muscular rigidity of the neck, which has not entirely cleared up. At the time of patient's admission retention of urine had replaced incontinence. Bladder was greatly distended and the urine in frightful condition. It was so loaded with pus that the catheter had to be withdrawn frequently and cleaned out. There is no tenderness over the bladder. He thinks he has lost about thirty pounds during the past eleven weeks. Bed-sores made their appearance about one week prior to his admission.

Physical Examination.—Patient is a young man of thirty years of age, poorly nourished and greatly emaciated. Emaciation is particularly marked in the lower extremities.

Eye Examination.—(See below.)

The mouth when opened is asymmetrical, being drawn to the right; this is not so noticeable when the mouth is shut. There is deviation of the tongue to the right. Tongue is slightly coated with a whitish fur.

Motor power in the left leg is entirely gone, and there is marked foot-drop on that side. He has the power of flexing both the thigh and leg on the right side. There is no foot-drop on the right side.

Reflexes.—Both knee-jerks are increased; more so on the left side. Plantar reflex is present on the right side; absent on the left.

Clonus is decided in left foot, to a less degree on the right side.

Patellar-clonus is present on the right side; absent on the left.

Sensation.—The right side of face, anterior half of right side of head, upper and lower thirds of right ear, right eyeball, right nostril, right half of tongue, gums, hard and soft palate, and arches on the right side, are either insensible to a pin-prick or else senation is dulled and much delayed. Owing to his long-standing catarrh, sense of smell cannot be taken.

The right nostril is constantly plugged up with mucous, bleeds readily to the touch and the membrane seems swollen. His catarrhal condition causes him some trouble on the left side, but it is not nearly so marked as on the right.

Sense of taste (tested with vinegar, oil of cloves and a solution of quinine) is gone on the anterior part of right side of tongue.

Eye Examination, by G. E. de Schweinitz.—Right Eye.—The cornea is crossed diagonally by an irregular band of infiltration, more dense in its centre, the result of a former keratitis. As this scar exactly crosses the pupil it is impossible to see the eye-ground. The iris moves freely; anterior chamber of normal depth.

V. = counting fingers at sixty centimetres. Left eye, no abnormalities. Disc and arteries normal, possibly some fullness of the retinal veins.

In the median line and in the right field of fixation there is homonymus lateral diplopia, due to paresis of right external rectus. The excursion of the eyeball in all directions, however, is perfect.

Ophthalmic Diagnosis.—1. Right corneal macula, result of former neuro-paralytic keratitis. 2. Paresis of right external rectus.

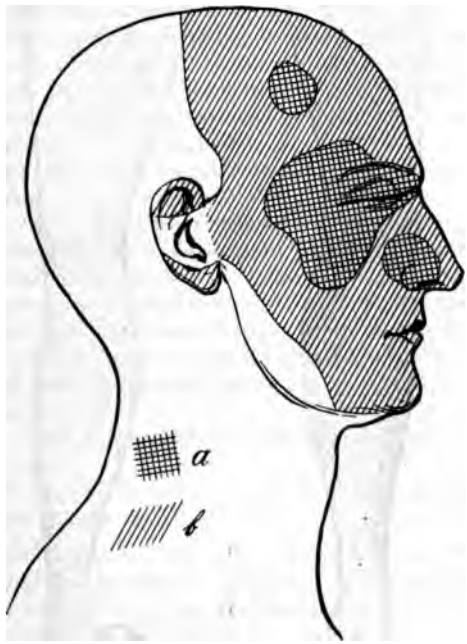
Both legs have areas in which sensation is delayed and blunted. The lower two-thirds of the thighs have delayed and blunted sensation. Sense of touch is much delayed on both lower extremities; in places it is absent. Except in one or two spots, sense of heat and cold seems lost from knees down.

The inner side of left thigh, which is less sensative to pain and touch, recognizes heat and cold, while the right does not. Sensation is sometimes delayed in upper extremities.

Hearing is equally good in both ears.

Pressure down the spine elicits decided tenderness over the sixth dorsal vertebra. There is also some tenderness over the vertebra immediately above and below.

FIG. 1.



Eyeball is absolutely anæsthetic. *a*. Sensation is gone. *b*. Sensation delayed or perverted. A pin-prick feels like head of pin was touching.

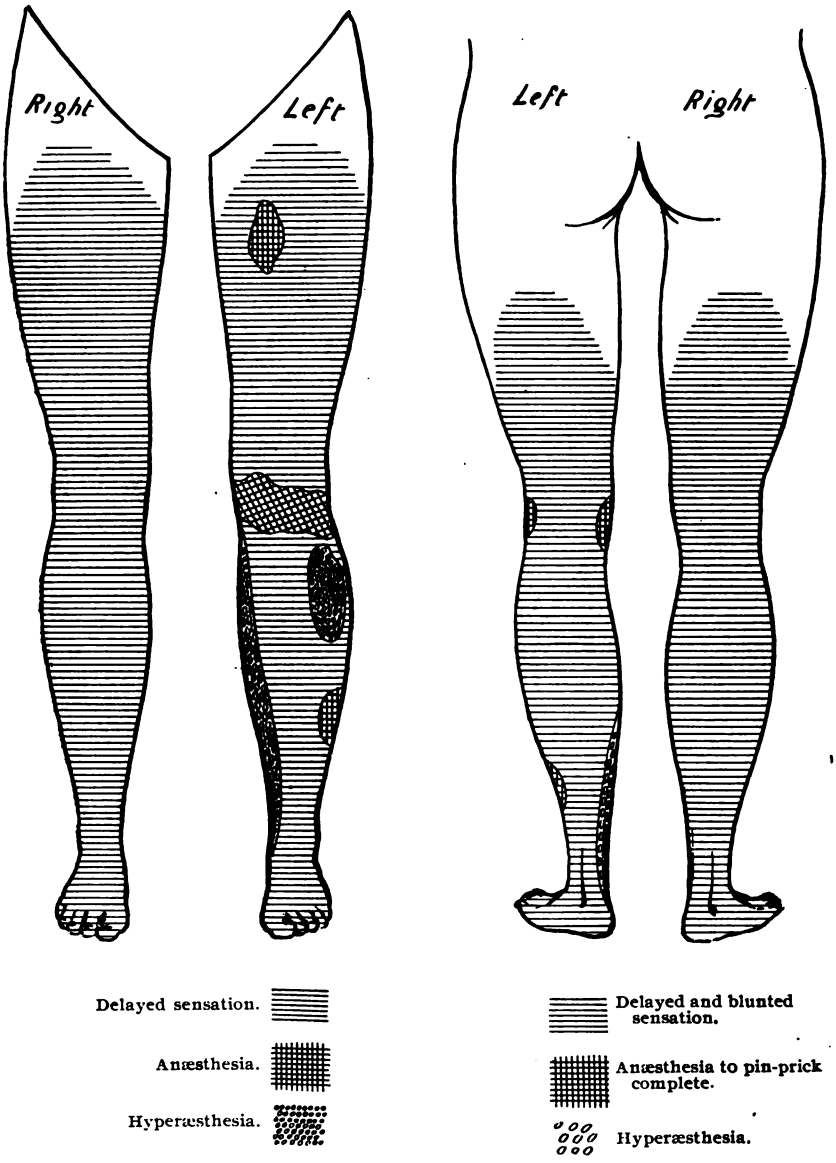
Lungs.—Negative.

Heart.—Sounds are not strong or forcible. Second sound is accentuated.

July 4, 1898.—Patient complains to-day of severe pain in his legs, the left especially. Pain resembles that of a toothache. In addition, he has girdle pains. The latter start about an inch from the spine on either side and radiate around the body between the pubis and umbilicus. He says it feels as though a red-hot cord was tied around his body. Bladder is irrigated twice daily. Urine is clearing up rapidly, pus being confined mostly to the last part.

Patient says he is troubled a good deal by involuntary contractions of the leg muscles. These contractions are confined to the flexors; both legs at times are

FIG. 2.



In the right leg the patient recognizes touch, but cannot tell whether the point or head of the pin touches. Sensation is also delayed. The left leg, except in areas, is like the right. On the left there are three areas of anæsthesia and two of hyperæsthesia.

violently flexed. There is considerable muscular irritability in the legs. Morphia had to be used several nights for the girdle pains.

July 13, 1898.—Bladder condition has improved. Partial paralysis of great toe on right foot has taken place during past few days.

Legs again tested for sensation. The left leg now has areas of hyperæsthesia. Sensation to a pin-prick feels like a knife was being thrust through the leg. The left tibia is excessively tender, the slightest taps with the finger causing severe pain.

July 16, 1898.—Patient had some slight incontinence of urine yesterday. During the night he complained of the right side of head and face feeling hot; nurse said it was very red. This morning there was marked unilateral sweating involving the anterior half of right side of scalp and the upper third of the face. It was very noticeable. Large drops of sweat were dotted over the forehead on the right; they extended to the middle line, where they abruptly ceased.

July 18, 1898.—The unilateral sweating before noted has extended downward and involved right side of neck and the anterior part of chest as low as sixth rib.

Yesterday, for first time, patient was able to use the left leg slightly.

July 24, 1898.—Ankle-clonus still present on both sides; more so on the left.

Knee-jerk on right side much exaggerated.

Knee-jerk on left side, which was at first markedly exaggerated, could not be obtained this morning.

Elbow-jerks and chin-jerk greatly increased.

Slight taps on the tibia still cause intense pain. Hyperæsthetic areas on left leg still remain. Contraction is evidently taking place in left leg; considerable rigidity is present when the attempt to straighten the legs is made.

Pains in the lower limbs have been frequent and severe for past few days.

Occasional injection of the conjunctiva of right eye has been noted. It lasts for only a few hours and then disappears, only to return in a day or so. Bladder condition remains about the same.

August 6, 1898.—Almost complete incontinence of urine now exists. Urine is clear and free from pus.

Examination of Urine.—Yellow; 1020; sediments, slight, acid; albumin, a faint trace; negative, microscopically.

Ethel Higbee, white, aged thirty-four years; birth-place, England; residence, Philadelphia; occupation, housework; admitted, Deecember 29, 1897.

Chief Complaint upon Admission.—Inability to use the legs well, pain in left hypochondrium, terrific headache and pain in eyeballs, swollen abdomen, incontinence of urine and fæces.

Family History.—Mother died, aged twenty-eight years, of pulmonary tuberculosis. Father died, aged fifty-four years, unknown cause. Maternal grandparents of good healthy stock. Paternal grandparents of rather delicate temperament. Patient "takes" more after the latter. Has only one brother, who still lives in vigorous health. A maternal uncle was subject to delirium tremens. No history of tuberculosis, other than given, or of insanity, obtainable.

Past Medical History.—Born in Leeds, England, in 1864; came to America in 1882; married in 1890. Was always a delicate child until her menstruation, at twelve years of age. Was always regular until marriage (having no dysmenorrhœa, small in amount and of a day's duration). After marriage it became irregular (after childbirth), painful, but only lasting one day. When a child, had measles, mumps, whooping-cough and chicken-pox. Later she had typhoid fever, aged

twenty-two years; "yellow jaunders," aged twelve years; influenza and pneumonia in Pennsylvania Hospital, for six weeks, March, 1894. Says she thought she had malaria five years ago. Has always been attended, since marriage, by a homœopathist. Had an eight months' child, which lived five days; was quite ill afterward, history resembling that of puerperal sepsis for some weeks afterward. There is a pretty definite history of syphilis obtainable. One month after marriage she noticed a small chancre or the left vulva (about size of finger-nail); it itched, gave no discharge, was single and to her knowledge not accompanied by bubo. She is positive that she neglected the chancre, not knowing its character, four about four months. As it was disappearing, a brownish, red, non-itching eruption appeared, universal in its distribution. She thinks it later became red and itching. For some time later her hair fell out, had severe sore throat, pains in "shin bones" and joints of severe character. Went to Delaware Breakwater and stayed two months, and with medicines cleared up disease.

History of Present Illness and Condition upon Admission.—Was apparently cured of her specific trouble by March, 1892, and had no trouble after that up to beginning of present illness, which first appeared in May, 1897. In the interim she did her own housework. In May, 1897, noticed a severe sore throat, which was treated successfully. At the same time she began to have failing sight; she had floating particles in front of eyes and objects seemed hazy. This lasted until September, when she began to feel unwell, drowsy, generally weak, chilliness and sweats. Following that, her legs began to fail, had "tingling" sensations and "crawling of ants" beginning on soles of feet. No *stroke*. All this time had severe headache, pain, and tenderness in spine (?), polyuria, and no incontinence, until just before admission, when both urine and fæces became incontinent. Patient is a medium-sized, well-built woman; no emaciation; countenance agreeable; skin, smooth; eyes, blue, and hair tinged with gray.

Physical Examination.—Spleen.—Extends in mid-axillary line from the eighth to eleventh ribs. Complains of some pain and tenderness in left hypochondrium.

Liver.—From the fifth rib to one-half inch above the costal margin in mid-clavicular line.

Lungs.—Inspection shows chest well formed, costal angle about 85°. Expansion normal and symmetrical. Scarcely imperceptible prominence of præcardium. Palpation shows anterior tactile fremitus, slightly more marked over the right upper lobe than over the left. Percussion shows lungs normally resonant.

Auscultation.—Vocal fremitus similar to tactile fremitus. No râles heard.

Heart.—Sounds, normal; no murmurs detected. Pulse full and regular.

Abdomen soft and flaccid; no tumorous mass palpable.

Legs are rather thin and spare. Inner plantar arch rather high. There are two scars the size of quarter dollars, and coalescing on the left tibia near the tubercle. Has free control in movements of shoulders, elbows and wrist and fingers, says fingers of right hand feel stiff. Elbow-jerks not present, sensation in legs and arms normal; also on chest and abdomen, excepting a narrow anæsthetic area on left chest, described below.

Knee-jerk is exaggerated perceptibly upon the left side and has a peculiar vibratory character (almost clonus-like). The right knee-jerk is not so pronounced and not clonus-like.

Ankle-clonus is markedly persistent and rhythmical on left side. Right indistinctly present. Toe-jerk not elicited on either side.

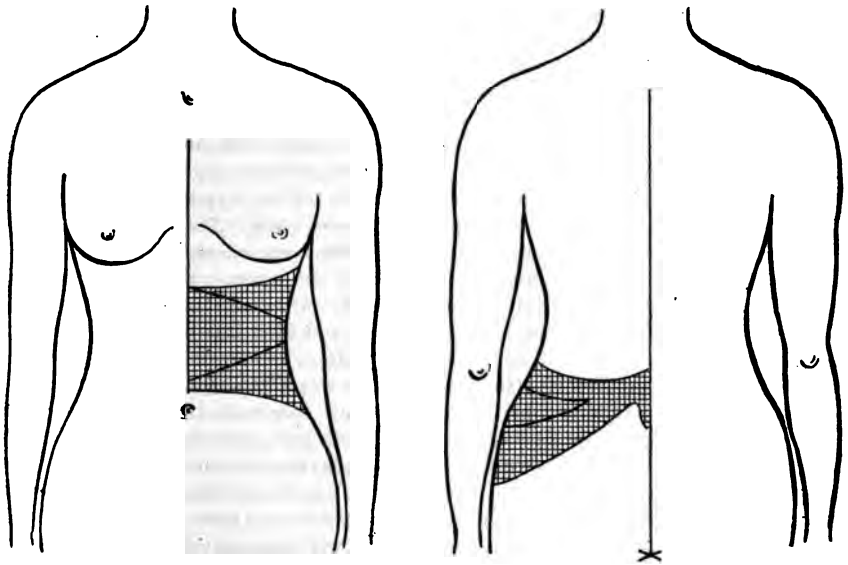
Plantar Reflex.—Marked on both sides.

Sensation.—There is anæsthesia to pin-prick on the left chest, extending from mid-sternum to spinous process of ninth dorsal vertebræ (which is tender on pressure), on an area representing the distribution of the ninth dorsal spinal (intercostal) nerve. This is distinctly anæsthetic, but there may a psychic element present in her responses to peripheral irritation when blindfolded. No other distinct anæsthetic area on body surface.

Ankle-clonus is markedly and persistently present on both sides, but most on left, it continuing with clock-like precision after removal of pressure.

Motion.—Patient has power to move both arms very freely; legs, however, with more difficulty; says they feel clumsy.

FIG. 3.



The shading shows the area anæsthetic to pin-prick. The heavier the shading, the more decided the anæsthesia. The wedged-shaped area marked > in the shaded area shows the part in which the sensation for heat and cold is lost.

Gait.—Supported, the patient walks with dragging of the feet, with most decided spasticity; would fall if not supported.

Eye Examination.—Left pupil is generally somewhat larger than the right. Left accommodates and responds to the light. Right responds to accommodation slightly, and even more sluggish to light.

Tongue held in median line and moved normally.

Speech not affected apparently.

Hearing—A small watch held about six inches from either ear can be distinguished with about the same ease.

Skin, except in places of scarring, is smooth, soft, pliable and normally moist. Has scarcely any abnormal sweating, or other vaso-motor disturbance. No bedsores or other trophic change.

Urine—Analysis, negative.

Notes on Progress of Case, January 14, 1898.—Yesterday patient had four dark-brown, liquid stools, with sharp, shooting, knife-like pains in the left hypochondriac region, worse on inspiration. Vomited slightly once last night. Was taken yesterday afternoon, while sitting up in a chair, with dizziness and shaking chill; says that the pain moved up into the neck and head. There was a dull, boring pain in the occiput, vertex and frontal regions of the head; eyes smarted and pained. Face flushed, eyes inflamed, and pulse though regular was rapid and full. Temperature was found to be 102⅘.° Urine voided and fæces evacuated involuntarily. Appetite poor. Slept fairly well last night, felt feverish, had great thirst.

To-day (*January 14, 1898*).—Feels better this A.M., is still weak, but headache is greatly alleviated. During the night had four large mucous, blood-streaked stools, and one small, white, mucous stool this morning. The tongue is coated with a brownish-white fur; has great thirst, but little appetite. Temperature 103⅘° at 4 A.M., down to 102⅕° at 10 A.M. Pulse rapid, about 128 to 140. Respirations about 28. Had a slight cough, which has disappeared; no expectoration. Pain in the side has disappeared, headache also has practically gone. Has tenderness on pressure and soreness in epigastrium.

March 7, 1898.—For some days after the re-administration of the K. I and bi-chloride of mercury (March 3, 1898), patient has improved wonderfully. The pain of which she complained in epigastrium and left hypochondrium is now quite gone, but there is still a sensation of numbness there. The bowels are to-day somewhat constipated, but continent. Has power over micturition; but, as she has done almost all the time since residence here, she voids it in large quantities. Appetite fairly good. Sleeps quite well. To-day, with one hand on a chair and one heavily on a fellow-patient, she was able to stand; her knees did not give way, as they no doubt would have on former occasions. Sometimes in the night she awakened perspiring freely. Her eyeballs now have free movement in the four directions (superiorly, inferiorly, internally, and externally), as they have for some weeks; headache not present much. Right arm, forearm, arm and fingers, thighs, legs and feet freely movable, although the legs feel heavy and the right arm has that sensation at times. The left arm has always been normal in every respect. Upon admission she had sensations of flashing, heat and cold, crawling of ants and heaviness (varying in their duration and intensity) in the legs, thighs, feet, right hand, forearm, arm, neck, head, between the shoulders and on the chest anteriorly, and on abdomen (says she once inquired if there were worms on her neck), but now these sensations have mostly disappeared. The left leg and thigh used to be weaker, now the right is. Tongue clean, and patient now appears cheerful and contented; is taking her treatment well, no gastric disturbance.

March 17, 1898.—Examination of the localized sensory symptoms by Dr. J. H. Lloyd. Patient has a distinct anæsthetic area on lateral aspect of the trunk on left side, somewhat ribbon-shaped, extending in the direction of the ribs, and almost in distribution of ninth dorsal nerve. At the ninth dorsal spine the patient winces on deep pressure, and says she suffers pain at that point. She complains also of a sense of burning and tingling along the course of this nerve in and below the anæsthetic area.

March 18, 1898.—With the aid of two patients, patient was able to walk farther and better than she had for many months. Says she walked about fifteen yards, but leaned heavily on her assistors, but had less control of right than of the left

leg, it having a tendency to turn or twist outward. The pain in the left side is not quite so severe as it was.

Eye Examination by Dr. George E. de Schweinitz.—January 7, 1898.—There is limitation of movement of right internus and of the right externus, but less marked. Upward movement is very slightly limited. Downward movement of the right eye markedly limited. Movement of the left eye normal in all directions. There is slight proptosis of right eye. The opthalmoscope shows perfectly healthy eye-grounds in right eye. The opthalmoscope shows perfectly healthy eye-grounds in the left eye, The optic nerves, vessels, etc., are perfectly normal. The right pupil is about 4 mm. in diameter. The left pupil is about 2 mm. in diameter.

Left Pupil reacts perfectly.

Right Pupil.—Direct and consensual reflex wanting.

March 8, 1898.—Eye Examination by Dr. Charles A. Oliver.—O. D.—Ophthalmoscope.—Pupil, oral; long axis, 95°; media, clear; disc, oval, 7° x 8°; long axis, 90°; scleral ring all around. Blackish conus in and out. Nerve gray in the deeper layers. Vessels in the long axis of the disc equal. Retinal arteries and veins normal in size.

O. S.—Pupil, round; media, clear; disc, oval, 7° x 8°. Long axis is 100°. Scleral ring all around. Trace of pigment beyond. Vessels at long axis of disc = 1 D. No gross changes in fundus. Retinal arteries and veins normal in size. Irides freely and equally movable to light, accommodation and convergence. Extra-ocular motion good in all directions, the former paresis being shown by a slight horizontal ataxic motion, which appears just as the utmost tension is relinquished. This being especially pronounced in the horizontal median, in lateral meridian, in lateral movements to the left. Associated with this is curvilinear motion down and in the right globe, during the movements to the left, showing a past paresis of the right superior oblique muscle.

April 16, 1898.—For the first time in nearly four months the patient is menstruating. The pain in left side of which the patient has complained is more troublesome. During the night vomited some. Seems to be very sensitive to action of bi-chloride when given hypodermically, it causing much pain, and in nearly every instance producing inflammation.

April 23, 1898.—Has been complaining of pain in left side of body and in left leg, so much so that she was unable to sleep well last night. To-day she says that the left leg feels heavy and is scarcely able to move it. The sensation to touch and pain is diminished on the left side.

April 26, 1898.—Patient is worse to-day. Had considerable trouble during the night. Has much burning pain in the left side. Her temperature has gone up to about 101° and has had few chilly sensations. This probably may be due to cellulitis of right arm set up by the hypodermic injection of bi-chloride of mercury (⅛ grain). The power of left leg is about the same, slightly diminished if any change.

July 18, 1898.—Patient requests her discharge, as her husband is to take her home. She has been improving steadily, and is now able to attend to all her personal wants. She can manage to get about very well with the aid of supports.

The old area of anæsthesia was gone over yesterday, and it was found to have broadened out greatly anteriorly. Instead of forming a ribbon-like band it is now wedge-shaped. Sensation to heat and cold is lost over this area anteriorly but not over back.

CASE III.—Edgar Miles, aged twenty-six years, white; occupation, teamster; residence, Philadelphia.

First Admission, July 28, 1896.—Diagnosis, cerebro-spinal syphilis; eloped, improved, April 20, 1897.

Notes on Case.—Patient cannot look inward or downward with either eye.

Trouble started two weeks ago, with diplopia and headache; throbbing pains in the head; nausea and vomiting. Station is good. Knee-jerk on left side absolutely gone. Knee-jerk on right side gone, but slight by reinforcement. Cremasteric-jerk present on both sides. Abdominal-jerk present on both sides. Biceps-jerk present, but not very marked.

August 12, 1896.—Complains of ringing and buzzing in left ear, and a tingling and twitching sensation in right arm. No loss of power; sensation unimpaired. For two weeks has been hard of hearing in same ear.

Examination of Urine.—Negative.

Temperature.—Normal.

Second Admission to Medical Wards, May 5, 1897.—Diagnosis, brain syphilis; Discharged, relieved, August 8, 1897.

Notes.—Chief Complaint.—Pain in bones of forearms, principally the left.

Family History.—Mother died of heart disease, aged sixty-one years; father died of consumption, aged seventy years. Two brothers alive, one of them a mute since two years of age, otherwise both well. Three sisters living, one of them has cancer of breast.

Past Medical History —Had the milder and severer diseases of childhood. Has had gonorrhœa six times. Six years ago had initial lesion of syphilis followed by secondary lesions in about six weeks. Underwent treatment with apparent cure. In June, 1896, noticed pupil of right eye larger than that of left. Strabismus followed. Had headache. Was treated in Blockley with recovery, and left the hospital about one month ago.

Present Illness.—Began about two months ago with difficulty in urination and incontinence, principally at night. Soon a dull pain developed in lower lumbar region. About two weeks ago, shooting pains and tenderness developed in bones of forearms; these pains are worse at night. Heart and lungs, negative; urine, negative; temperature, normal. Uses tobacco moderately; alcohol, occasionally to excess.

Third Admission (August 25, 1897) to Nervous Wards.—Diagnosis, hemianopsia; eloped, August 4, 1898, relieved.

Chief Complaint.—Pain in back of head and failing vision.

Present Illness.—About two weeks ago (August 10th), noticed that everything appeared to be dark, and had pains in the head. The pain appears to be localized in the occipital region to the right of the median line. He also has pain in the right eye; pain is sharp and shooting; worse at night. Vision has been growing worse since he first noticed the pains. When looking ahead, says that the side fields of vision appear black.

Patient is tall, slight and anæmic. Pupils, equal; eyes react slightly to light, but readily to accommodation. Left side slightly weaker than right. Left dynamometer, 25; right dynamometer, 40.

Deep reflexes slightly increased. No ankle-clonus.

August 30, 1897.—Three days ago patient walked well. Forty-eight hours ago bowels became loose; was put to bed; no fever. Now complains that right leg is numb, also in ring finger of left hand. Left arm is weaker than before. No diplopia now.

Examination.—Has grown very much weaker, can scarcely stand. Can move legs well in bed, but the left leg cannot be raised against moderately slight resistance. Can move right arm well. Movements of left arm are paretic. There is marked wasting of the fingers and forearm of left side; some wasting of upper arm and shoulder. No wasting of right arm or either leg. The paresis of left arm has increased in the last two days.

The knee-jerk is active; left is exaggerated. Plantar-jerk active on both sides. Right biceps-jerk, normal; left, exaggerated. Muscle-jerk in arms present. Chin-jerk present. Sensation to touch normal over entire body. No palsy of face. Tongue protrudes straight.

Eyes.—Pupils equal, do not react to light; do to accommodation.

Patient is blind on nasal side of both retinæ. Cannot turn either eye completely to the left.

September 1, 1897.—Left lower face is markedly paretic; upper left face slightly paretic. Water dribbles.

September 3, 1897.—No hemianopsia. Says he sees clearly across the room. Right pupil is much larger than the left. Some power in left arm and leg. Has control of bladder and rectum, first time in several days.

September 5. 1897.—No power over rectum this A. M. Pupils react to light and accommodation.

September 6, 1897.—Left pupil largest. Moderate dilatation.

Examination, by Dr. C. A. Oliver.—O. D.—Pupil, round; media, clear; disc, oval, 6 x 7; long axis, 90°. Scleral, red all around, traces of conus in and out. No gross changes in fundus. Best seen by 1 D.

O. S.—Pupil, round; media, clear; disc, oval, 7 x 8; long axis, 90°. Scleral, red all around. Broad blackish conus in and out. Long axis, disc = 1 D. No gross changes in fundus.

When light is thrown on left eye it does not react till light is brought in front, and to nasal side. Same is true of right eye, thus proving Wernicke's sign.

Field of Vision, roughly taken with Ophthalmoscope.—Handle showed temporal hemianopsia of right eye with diminished field vision of left eye. More diminution on nasal side, thus showing tendency to right homonymus hemianopsia. In view of shifting fields from bitemporal hemianopsia with Wernicke's sign, due to lesion in anterior or posterior fork of commissure. Is now shifting to left tract, giving a right homonymus hemianopsia.

September 8, 1897.—All paralyses about same. Right pupil, widely dilated; lert, normal. Marked weakness of left side on walking. Gaining use of left arm.

September 9, 1897.—This A. M., left pupil is fully twice the size of right. Tongue heavily coated with a greenish-brown material. Complete power over bladder; partial over rectum. Says sight is improving.

September 11, 1897.—At 9 A. M., right pupil dilated twice the size of left; 10.30 A. M., left, dilated some; 11.45 A. M., pupils equal; 6.30 P. M., right again dilated; left, normal. Paralysis of the face distinct. Other palsies clearing up nicely.

September 17, 1897.—Pupils equal. Left side of face drawn to right. Left arm and leg improving. Patient is walking about ward.

September 23, 1897.—Friday, patient has a distinct left hemianopsia. Paralysis is clearing up in both arms and legs.

August 4, 1898.—Patient eloped this afternoon.

below the summit of the cornea. Generally a small conjunctival flap was allowed to remain. The same character of flap was usually made after preliminary iridectomy.

In the cases submitted to simple extraction the method employed by Dr. Knapp was practised, that is to say, the corneal section comprised exactly the upper half of the cornea, or sometimes a little less, the section passing in its whole extent exactly through the transparent margin of the cornea, the knife remaining in the same plane throughout. In many cases a small, central conjunctival flap was formed, which in my opinion is, if anything, an advantage.

The linear extractions were performed in the usual manner with a keratome, which was entered about one mm. within the margin of the cornea and passed onward until it made a wound about five mm. wide. A sharp cystotome was then introduced and the capsule of the lens freely lacerated, the soft lens-matter being made to extrude by counter-pressure on the cornea with a horn spud, while the outer lip of the corneal wound was depressed with a curette.

The capsule was practically always opened by an incision shaped like the letter " T," the transverse cut being above. In three instances the lens was extracted in its capsule.

Operations designed to ripen the cataract were performed only twice.

For several days before operation the patient's face, eyelids and ciliary margins are frequently bathed with soap and water. The nares are sprayed with equal parts of listerine and Dobell's solution, or with peroxide of hydrogen suitably diluted, or, in recent times, following the recommendation of Dr. J. A. Lippincott, with a solution of permanganate of potassium (1 : 5000). Immediately preceding the operation—*i. e.*, an hour before it begins—the face, the closed lids, eyebrows, brow and scalp border are treated first with soap and water, then with alcohol and finally with corrosive sublimate (1 : 2000); none of these substances are allowed to enter the conjunctival sac. The ciliary margins are carefully cleansed with soap and water, and a 1 : 5000 bichloride solution. A compress soaked in a similar bichloride solution is placed over the eyes until they are opened for the cleansing of the conjunctival cul-de-sac just before the section is made. This cleansing is done with tepid boric-acid solution, introduced with some force so as to flush out the entire sac. The local anæsthetic, in all

instances cocaine 4 per cent., sterilized by boiling, is used three times, at intervals of about five minutes. Just before making the puncture the cornea is wiped with a small pledget of cotton steeped in boric acid or physiological salt solution. The instruments, wrapped in cotton, are sterilized by boiling, then transferred to a dish of alcohol, and then laid in a tray filled with sterilized water, from which they are taken by the assistant in the order the operator requires.

Both eyes are closed after operation with a light dressing, consisting of an oval piece of gauze, soaked in boric acid, a small pad of absorbent cotton and a few turns of a gauze roller. If all goes well, the unoperated eye is released on the third or fourth day, and all bandages are removed at the end of a week.

Complications During and After the Operation—The complications which were noted as occurring during the operation were: Dislocation of the nucleus of a hypermature cataract into the vitreous, twice; unusual collapse of the cornea, once; loss of vitreous, seven times; very unruly behavior of insane patients, twice.

The complications noted as occurring after the operation were the following: Iritis, twice; irido-cyclitis with closure of the pupil, twice; prolapse of the iris, after simple extraction, three times; prolapse of the iris, in the angle of the coloboma after combined extraction, three times; hyphæma, twice; traumatic, once, and once spontaneous and recurring; striped keratitis, twice; localized infection of the flap with iritis, once; glaucoma, once; post-operative insanity, once; slow closure of the wound, twice; intra-ocular hemorrhage, twice. In one case there was sudden death at the end of forty-eight hours, preceded by suppression of urine in a woman suffering from Bright's disease.

Of the three cases of prolapse of the iris which occurred with the simple extractions, the prolapsed iris was replaced on the sixth day by the use of eserine, in one instance; it was abscised in another, while in the third it was treated expectantly, that is, with pressure bandage, etc., but resulted in irido-cyclitis with closure of the pupil. In one of the cases of prolapsed iris which occurred in the combined extractions there was mild iritis.

With the exception of the two cases of intra-ocular hemorrhage, the localized infection of the flap and the case of irido-cylitis just quoted, the ultimate result was good in spite of the complications which have been enumerated.

Several of the cases belong to the complicated list which have been reported *in extenso*, and from which by the very nature of things it would not have been possible to have obtained more than an operative success. Some of these complications were of such an interesting nature that they are reserved for a more extended notice, which see.

In sixty-six instances there was normal healing, that is, the period from the time of operation to complete recovery was uneventful.

Secondary Operations.—These were as follows: Discission, that is, laceration of the capsule, twenty-six times; Agnew's capsule operation, once; abscission of prolapsed iris and stitching of the wound, once; iridotomy, once; irido-cystectomy, twice. The result of these operations in all instances was favorable and no complications occurred.

Visual Results.—Inasmuch as the vision was tested sometimes at twenty feet, sometimes at six metres and sometimes at five metres, it has been expressed in the following table. The complicated cases are included in a separate table on page 68.

1 = 20/XX	= $\frac{6}{6}$	= $\frac{5}{5}$	= 12	cases
$\frac{2}{3}$ = 20/XXX	= $\frac{6}{9}$		= 5	"
$\frac{1}{2}$ = 20/XL	= $\frac{6}{12}$	= $\frac{5}{10}$	= 9	"
$\frac{2}{5}$ = 20/L	= $\frac{6}{15}$		= 9	"
$\frac{1}{3}$ = 20/LX	= $\frac{6}{20}$	= $\frac{5}{15}$	= 8	"
$\frac{2}{7}$ = 20/LXX	= $\frac{6}{22}$		= 5	"
$\frac{1}{4}$ = 20/LXXX	= $\frac{6}{25}$	= $\frac{5}{20}$	= 3	"
$\frac{1}{5}$ = 20/C	= $\frac{6}{30}$	= $\frac{5}{25}$	= 6	"
$\frac{1}{8}$ = 20/CLX	= $\frac{6}{50}$	= $\frac{5}{40}$	= 4	"
$\frac{1}{9}$ = 20/CLXXX		= $\frac{5}{45}$	= 1	"
$\frac{1}{10}$ = 20/CC	= $\frac{6}{60}$	= $\frac{5}{50}$	= 7	"
Good[1]			= 1	"
$\frac{1}{37}$ = 4/CXLVIII			= 1	"
Counts fingers			= 1	"
Light perception			= 2	"
$\frac{1}{\infty}$			= 1	"

This may be summarized as follows:

Success, 70 cases, or 93.3 per cent.
Moderate success, 1 case, or 1.3 per cent.
Failure, 4 cases, or 5.4 per cent.

In one case sudden death occurred forty-eight hours after a perfectly smooth combined extraction; autopsy was forbidden, but the symptoms were those of chronic Bright's disease with suppression of urine.

[1] See No. 74, Table of Cases.

It will be noted that examination of the tables show in thirty-six instances the last recorded vision was tested with a simple spherical lens, that is, the indications are that no attempt was made to correct any co-existing astigmatism. Doubtless if this had been done the visual results in some of these instances would have been higher than those which are recorded. From the very nature of the cases, however, it was often difficult to order a cylindrical glass, and, in fact, it is not certain whether many of the patients would have appreciated the refinement in vision which such correction would have given.

The moderate success, and one of the failures, both cases of iridocyclitis with closed pupil, but with perfectly good light projection, could certainly have been converted into visual success by iridotomy or iridectomy, but both patients declined to allow operative interference.

The case histories of the failures were as follows:

CASE I.—Sarah Ann, aged eighty-nine years, a senile dement suffering from chronic bronchitis and conjunctival hyperæmia, had removed from her left eye a mature cataract, and during the operation was exceeding unruly. Infection of the flap was evident at the end of twenty-four hours. This infection remained localized, and the eye healed with a large leucoma, the ultimate vision apparently being light perception. Almost from the moment of the operation the patient was delirious and frequently pulled off her bandages. Therefore, it was difficult to determine whether the infection was caused by the contact of her fingers with the eye, or whether the infection had come from the inflamed conjunctiva, or through the tear-ducts from the naso-pharynx. (See No. 13, Table of Cases.)

CASE II.—This case has been reported in the *Annals of Ophthalmology and Otology*, Vol. III, 1894, from which the following account is taken: (See No. 26, Table of Cases.)

Agnes Donovan, aged sixty-five, was admitted to the ophthalmic wards of the Philadelphia Hospital, June 23, 1892. Her right eye was sightless and shrunken, the result of an injury inflicted many years ago. In the left eye there was a dense nuclear opacity in the crystalline lens, the surrounding portions of this structure being clear and transmitting the reflex from the fundus oculi. Vision equalled counting fingers. The eyeball was prominent, of type usually seen in myopic refraction; the anterior chamber deep; the conjunctiva slightly congested, and the anterior perforating vessels more tortuous than is normal.

She was not seen again until nearly one year later, May 15, 1893, when the appearances already described remained unchanged. The general health of the patient was good; there was neither albumin nor sugar in the urine; but the temporal arteries were hard and the pulse characteristic of high arterial tension. On the 17th of the same month preliminary iridectomy was performed—a perfectly smooth operation—and the capsule of the lens stroked with a spatula. There was no complication in the healing,

One month later, June 25, 1893, the entire lens was opaque; the eye was quiet,

having, to use Mr. Carter's apt expression, entirely "forgotten the iridectomy;" the iris was mobile, and, so far as loupe examination showed, not atrophic, the light projection perfect and the tension normal. Extraction was performed by the 3 mm. flap operation, the knife being entered exactly at the corneo-scleral junction, at the outer extremity of a horizontal line which would pass 3 mm. below the summit of the cornea. Immediately at the completion of the section, and before the cystotome was used, a bead of vitreous presented in the wound, the crystalline lens being tipped somewhat upward. The cataract was quickly, but with some difficulty, removed with a scoop, its exit being followed by a quick gush of vitreous. For an appreciable length of time there was slight diminution in the tension of the eyeball, followed quickly by a restoration of the tension to the extent of an unusual filling out of the ball. The flap fell nicely into place, the margins of the coloboma were clear, the vitreous ceased to escape and the fingers could be counted. The usual light antiseptic dressing was applied, and the patient, who had been operated upon in the bed in which she was to remain, was moved to a darkened corner of the ward.

She complained almost at once of a sickening pain in the back of the head and of nausea. Suspecting that I was in the presence of the gravest of all accidents which may occur after cataract extraction, a hypodermic injection of morphia, one-sixth of a grain, was administered, and the patient's head was raised above the horizontal level to the height of three pillows piled one above the other. In ten minutes, blood began to ooze through the bandages. The dressings were quickly removed and the palpebral fissure found distended with a large blood-clot, while fluid blood flowed freely down the cheek. Remembering the advice of Knapp, the blood-clots were carefully removed, one from its position between the lids and another which protruded through the corneal incision. The conjunctival cul-de-sac, the wound and the surrounding area were carefully cleansed with a solution of bi-chloride of mercury, 1 : 5000, and a full, firm antiseptic dressing was applied. The patient was required to sit bolt upright in the bed, properly supported with pillows. After another one-sixth of a grain of morphia was administered, the sickening pain in the back of the head disappeared, and she expressed herself as feeling comfortable.

Two hours later I was hastily summoned by the house surgeon with the statement that the bleeding had recommenced. Precisely the same treatment which has been detailed was carried out, although no additional morphia was administered until later in the night, when some return of pain caused the resident surgeon to repeat the dose. At the expiration of twenty-four hours the bandages were removed, and, with the exception of a little blood-staining on the pad next the eye, there had been no renewal of hemorrhage. The cornea was perfectly clear; a blood-clot (which was removed) separated the lips of the wound and partially filled the remains of the anterior chamber, and there was distinct light perception in the lower and outer part of the field.

The subsequent history of this case may be dismissed in a few words: The hemorrhage did not recur, and panophthalmitis did not supervene. Gradually the eyeball has shrunken, although it still preserves its form, being puckered around the corneal margins. The cornea remains clear, although prominent and small in area, somewhat resembling a pellucid staphyloma, but the faint light perception, which lasted for about forty-eight hours, has entirely disappeared and the eye is totally blind.

CASE III.—Rosina Schmidt, aged seventy years, in good health, and with normal ocular conditions, had removed from her right eye a senile cataract by simple extraction. On the fourth day there was prolapse of the iris, which was treated expectantly, that is, by pressure bandage, etc. After the inflammatory process had subsided, the patient was able to see the watch face with a + 10ᵈ, but six months later she was again seen, and the pupil was then drawn upward and occluded by a dense, web-like membrane—the end stage of irido-cyclitis. There is no doubt, inasmuch as the light projection was perfect and the patient could count fingers at a few inches, that either iridotomy or iridectomy would have restored good vision, but she positively declined to permit operative interference, although, curiously enough, she was willing to submit to an extraction of the cataract upon the opposite side and secured a vision in this eye of 6/9.

CASE IV.—Julius Wolf, aged seventy-nine years, a mildly-demented patient, suffering from chronic Bright's disease and typical widespread angio-sclerosis, had an over-ripe subluxated cataractous lens of the right eye. The iris was tremulous, and there was slight chronic conjunctivitis. The usual corneal section was made, comprising nearly the upper half of the cornea. When the capsule was incised, the soft cortex was easily delivered, but the nucleus slipped backward and upward. Several attempts to dislodge it through the circular pupil proved ineffectual, and, therefore, a small iridectomy was performed in the hope that through this artificial opening the delivery of this nucleus would be facilitated. By this time, however, the nucleus had disappeared into the well-nigh fluid vitreous which was escaping. Several attempts with loupe were ineffectual, as were also those efforts to bring the nucleus to the lips of the wound by direct pressure upon the eyeball.

Further attempts did not seem justifiable, which was also the opinion of the confrère who was watching the operation, and it was determined to allow the nucleus to remain, as after a couching operation. The lips of the wound were easily coapted, the pupil was black and the patient readily counted fingers. No disturbance occurred until five hours later, when, after a severe pain, there was a sharp intra-ocular hemorrhage which burst open the wound and thoroughly soaked the bandages. These were removed, the gaping wound was freed from blood-clots, and it was noted that the hemorrhage apparently came from the ciliary region. Among the blood-clots was found the small nucleus. The lips of the wound were approximated with three sutures and the pressure bandage reapplied. Slight oozing of blood occurred for a few days, staining the pad, but there was not again a free hemorrhage. No decided reaction occurred, the cornea remained clear, the anterior chamber refilled, and the iris could be plainly seen, although it was somewhat discolored. The coloboma was filled with a reddish mass, probably the remains of a blood-clot. The patient's general condition was very bad. He passed into a semi-comatose state with involuntary passages of urine and fæces. Sometimes for a day or two he would rally, and occasionally would have one or two days in which he was quite lucid and cheerful. On these days a number of tests indicated the presence of good light projection in all portions of the field. Death occurred from uræmic coma about six weeks after the operation.

Once before I have seen the nucleus of a Morgagnian cataract dislocated into the vitreous and the eye heal without reaction, the patient having very good vision, which was still present six months after

operation. This patient lost her eye subsequently from another operation, probably a discission, at the hands of another surgeon, the details of which I do not know. (See No. 8, Table of Cases.) It was this experience that led me to think it would be better to allow the escaped nucleus to remain in the vitreous than to make further attempts to dislodge it, which would almost certainly have evacuated the eyeball on account of the fluid condition of the vitreous. In the light of subsequent events, this may be regarded as a mistake in surgical judgment, although I still think that, if I had persisted in my attempts to recover this nucleus, the eye would have been lost.

The visual results of the complicated cases were as follows:

10/cc	= one case.
6/c	= one case.
D 45 at one metre	= one case.
Sn. cc at six inches	= one case.
Counts fingers	= three cases.
Light perception	= three cases.

The following are brief case histories of the complicated cataracts which were removed from eyes so diseased that the visual results, although in some instances surprising, could not, from the nature of things, reach even the lowest average of visual success after cataract extraction:

CASE I.—Charles Lewis, colored, aged forty-one years, suffered from constitutional syphilis and iritis, which produced secondary glaucoma with iris bombè. One year prior to his admission to the hospital an iridectomy had been performed by another surgeon. There was light perception in the lower and outer portion of the field. After extraction of the lens there was normal healing, and the ophthalmoscope revealed a deeply-cupped, atrophic optic nerve. The ultimate vision was counting fingers in the lower and outer field. (See No. 3, Table of Cases.)

CASE II.—James McMahon, aged fifty-eight years, a chronic alcoholic with albuminuria, had an immature cataract in his left eye with imperfect light perception. The lens was extracted after preliminary iridectomy and three discissions performed at different intervals to cause absorption of the remaining cortex. A perfectly clear coloboma resulted, and the ophthalmoscope revealed a small, entirely atrophic disc with shrunken arteries. The ultimate vision was counting fingers and the patient was able to walk about unaided. (See No. 7, Table of Cases.)

CASE III.—Emmanuel Mack, aged seventy-five years, had a large corneal macula upon the left eye, the result of an extensive ulcer in childhood. After a preliminary iridectomy, the lens was extracted and absorption of the cortical remnants produced by two discissions, followed later by Agnew's capsule operation. In addition to the corneal macula, the ophthalmoscope revealed huge posterior staphyloma and disseminated choroiditis. The ultimate vision was 10/cc. (See No. 12, Table of Cases.)

CASE IV.—John Duffy, aged seventy-nine years, an insane patient, and at times subject to explosions of insanity, but at the time of operation believed to be sufficiently quiet to remove a mature senile cataract of the right eye. During the operation—in fact, while the knife was still crossing the anterior chamber—this patient became absolutely uncontrollable, fighting and striking at the operator, so that it was impossible for the assistants to hold him. In spite of this, the lens was delivered, but with a large loss of vitreous. Healing was perfectly kind, but a whitish membrane could be seen behind the pupil, probably a detachment of the retina. The patient's condition was such that accurate examination was impossible. Before dismissal from the ward the ultimate vision appeared to be counting fingers. He was seen one year later with the eye in the same condition, but apparently the vision not more than light perception. (See No. 24, Table of Cases.)

CASE V.—Samuel Collins, colored, aged thirty-nine years, a sufferer from phthisis, had a large adherent leucoma following an ulcer of the right eye. There was faint light perception in the lower and outer field, and no hope of much improvement in vision was held out to him, although it was deemed proper to attempt a removal of the lens, which was done by linear extraction after division of the anterior synechia. The recovery was complicated with iritis, and the ultimate vision the same as that before operation, namely, light perception in the lower outer field. Three months later, it is stated that the eye became irritable and was enucleated by another surgeon. The history at this time was not obtainable. (See No. 31, Table of Cases.)

CASE VI.—John Elliott, aged forty years, in feeble health, had milky-white nuclear cataracts and doubtful light perception, except above. The tension was minus, and the functional examination of the eye indicated detachment of the retina. Iridectomy had been performed five years previously by another surgeon. Following the extraction of the lens there was a free escape of thin vitreous, which continued to ooze for five days, when the wound closed. The ultimate vision was light perception in the same portion of the field as it had existed prior to the extraction. (See No. 32, Table of Cases.)

CASE VII.—Thomas Morrow, aged fifty years, a melancholic, had secondary glaucoma following iritis, with complete obliteration of the nasal field, that is to say, there was light perception only upon the temporal side. After preliminary iridectomy, extraction of the lens was performed and still later iridotomy. The ultimate vision was 1/45. (See No. 64, Table of Cases.)

CASE VIII.—John Rohr, aged fifty-four years, had always been myopic and had a semi-luxated calcareous lens with doubtful light perception. After extraction of the dislocated lens, aided by preliminary iridectomy, the eye-ground was seen to be the seat of most extensive atrophic retino-choroiditis and atrophy of the disc. The ultimate vision was shadows. (See No. 67, Table of Cases.)

CASE IX.—S. Desango, a melancholic, had suffered from iritis which produced cataract. The functional examination of the eye indicated detachment of the retina. Following the extraction, there were successive attacks of hyphæma, which, however, ultimately subsided, and, when last seen, the patient was able to walk around unaided and could distinguish Snellen 200 when held close to the eye somewhat eccentrically. (See No. 73, Table of Cases.)

CASE X.—Andrew Stenger, a patient in good health, had central corneal macula, somewhat horseshoe-shaped. After preliminary iridectomy, the cataract was extracted and the healing uneventful. A rough test of vision showed about 2/30,

with which the patient was well satisfied, and which, considering the condition of the cornea, was very good. It is possible that an iridectomy placed below and outward might have increased this vision, but as the patient had had a successful extraction of cataract upon the opposite side, with good reading vision, this did not seem necessary. (See No. 81, Table of Cases.)

In addition to the histories of the complicated cases and the failures which have been recorded, the following somewhat more detailed records would seem worthy of publication, although the patients have not been included in the complicated list :

CASE I.—*Spontaneous Dislocation of Both Crystalline Lenses into the Anterior Chamber ; Complete Blindness of the Right Eye ; Successful Extraction of the Left Lens.*[1]

Ellen C., aged forty-eight years, married when she was twenty-eight years old, and had one child born seven years later. The patient, except for the ordinary exanthemata of childhood, had never been sick in bed, had worked hard all her life at washing, and had been occasionally troubled with rheumatic pains in the shoulders and limbs. From early childhood she was near-sighted ; her mother was also myopic ; her father, two brothers, and one sister had good sight. Five years ago, while walking along the street during a thunder-shower, a sudden flash of lightning greatly startled her ; immediately afterwards phosphenes appeared before her eyes, chiefly in the form of round-colored rings, succeeded in a short time by "a scum which gradually grew thicker" until sight was obliterated, and her husband noted "a white substance in front of her eye." The eye was painful, red and watery, but gradually quited down and ceased to be troublesome, although no sight returned to it. The left eye had served her a useful purpose, except for the inconvenience of myopia, until about six months ago, when the vision distinctly depreciated, and two weeks before her admission to the hospital, while stooping over to tie her shoe, and suddenly raising her head, the sight was instantly extinguished. She came to the hospital January 30, 1889, and the following conditions were revealed by the examination :

Right Eye.—A large eyeball, tension + 2 ; coarse injection of the posterior conjunctival vessels with distention of the anterior perforating arteries ; the entire cornea roughened and hazy, except a small margin above, which was still transparent. Upon turning the eye strongly downward the somewhat shrunken and opaque lens was seen lying in the bottom of the anterior chamber ; no view of the deeper structures of the eye was possible.

Left Eye.—The eyeball was prominent, the conjunctival and episcleral vessels were densely injected, while scalding tears bathed the surface of the globe, and severe pain shot through it and the brow and temple. The cornea was transparent, and lying in the bottom of the anterior chamber against the posterior surface of the cornea the lens was clearly seen.

The patient was put to bed, directed to lie on her back, and cocaine and atropia were instilled into the eyes. On the following morning both lenses had slipped

[1] This case has been reported in detail. See *University Medical Magazine*, Vol. II, 1889–1890, p. 84. The case abstract given above is taken from this report.

back into the vitreous chambers, and sight sufficient to discern the objects in the room was restored to the left eye, which presented in its interior, as far as such examination was possible, the changes incident to a high myopia. A few days later, after permission had been given to leave her bed, and while performing some slight ward work, the lenses again appeared in the anterior chambers. Again the patient was put to bed on her back, with the result that, in a few hours, they returned into the vitreous bodies. A third time, after getting out of bed and doing a little sweeping, the lenses reappeared in the anterior chambers. An operation was now suggested to the patient, to which she consented on the condition that ether should be administered. Accordingly, after the instillation of a solution of eserine in the hope of contracting the iris behind the lens, and thus forbidding its disappearance into the vitreous humor, she was seated upright in bed, propped up by a number of pillows, her head bent forward, and the anæsthetic administered in this position. She fortunately took the ether most kindly, without struggling, and when fully anæsthetized, an incision was made with a Graefe cataract knife in the lower corneo-scleral junction and an attempt made to deliver the lens with a Daviel spoon. This not proving quickly successful, a wire loop was substituted and introduced behind the lens, which had in the meantime turned slightly on its axis and partially protruded backward through the pupillary space, and it was delivered safely with the loss of only a few drops of vitreous. Scrupulous antiseptic precautions were followed during the operation. No vomiting occurred after the ether, and a compress bandage, which had at first been adjusted with unusual firmness, but was in a few hours loosened, was applied, and a dose of morphia and bromide of potash administered.

During the night the patient complained somewhat of pain in the head, of a shooting character, but on the following day was quiet, and an examination of the eye externally showed no swelling or apparent reaction. The case went on to good healing with one troublesome complication—an aggravated spasmodic entropium. This, together with the pain which was constantly present in the blind eye of the opposite side, led to an enucleation of the latter, and to the removal of a strip of skin and orbicularis muscle from the lower lid of the former, with the result of entirely relieving the inversion and the constant lachrymation. After all redness had passed away, an ophthalmoscopic examination showed an irregularly oval disc surrounded by a huge atrophic conus, and scattered through the eye-ground numerous patches of choroido-retinitis. In spite of this the sight was useful, 10/c, the removal of the lens having apparently neutralized the former myopia, as no glass increased the acuity of vision. (See No. 4, Table of Cases.)

CASE II.—*Gumma of the Iris, Producing Cataract; Preliminary Iridectomy followed by Extraction; Glaucoma; Recovery with Excellent Vision.*[1]

Although this case has been reported in full, it contains certain interesting features, which seem to render proper the reproduction of its salient features in the present place.

Robert Kingsberry was admitted to the Ophthalmic Wards of the Philadelphia Hospital on the 28th of December, 1891, presenting the following symptoms: In

[1] This case has been reported in full in *Annals of Ophthalmology*, Vol. II, 1893, p. 143.

the right eye the cornea was hazy, and a large, yellowish-white mass, not crossed by vessels, which pushed its way toward the ciliary border, partially filled the anterior chamber, at the bottom of which a layer of pus had collected. The other eye was similarly affected, the deposit growing from the iris having a whitish rather than a yellowish-white appearance, and also a tendency to spread toward the ciliary region. There were violent photophobia, chemotic conjuctiva and swollen lids.

Shortly after admission, the patient exhibited a curious type of delirium, characterized by a fixed delusion of persecution, terminating in an irregular type of delirium tremens. The mental condition of the patient was such that an exact clinical history was not obtained, nor was the date of syphilitic infection, which, however, was undoubted, accurately determined. It probably occurred about two years before the gummata appeared in the iris.

After the usual treatment for such condition, the violence of the inflammation gradually subsided, the eyes became quiet, but the irides were atrophic and well-marked scars were visible at the spots formerly occupied by the gummatous deposits, both pupilliary spaces being partially occluded.

He remained in the hospital for some time, and then disappeared, returning again in the fall of 1892. When he was examined, November 1, 1892, the pupil of the right eye was irregular, partly occluded with lymph and iris pigment, a broad scar being visible in the iris to the nasal side; ophthalmoscopic examination was impossible. Vision equalled counting fingers at one foot. In the left eye there was a similarly scarred iris, a somewhat larger pupil, behind which a white cataract was visible. The light perception was good in all parts of the field.

About two weeks before, namely, on the 16th and 20th of October, my colleague, Dr. Gould, had made two discissons, without, however, causing perceptible absorption of the cataractous lens. Dr. Gould very kindly referred the case to me when I came on duty, the first of November, for further operative interference. As two well-considered and carefully-performed discissions had been practically without effect, it was deemed advisable that a formal extraction should be undertaken. On November 7th, I performed a small, upward iridectomy, obtaining a clear coloboma, save a slight irregularity at the lower margin, marking the position where the iris had been attached to the capsule of the lens. The healing was perfectly smooth, and on the 28th of the same month the lens was extracted without difficulty, except that the capsule was found exceedingly tough. No accident occurred during the healing, and on the fifth day after the operation the bandages were removed and the patient allowed the liberty of the ward, with the protection of a shade.

On December 13th, the patient began to complain of pain in the eye, which became watery, and a small spot of infiltration appeared in the lower margin of the cornea. Atropine was instilled for one day at intervals of four hours, but on the following day he was found in a pitiable plight, complaining of intense pain through the entire left side of the head. The lids were œdematous, the conjunctiva slightly chemotic, the cornea hazy throughout its entire surface, and the T. + 2. Vision amounted to faint light perception.

He was freely leeched from the temple; eserine drops, one-half grain to the ounce, together with a two-per-cent. solution of cocaine, were instilled into the eye every two hours, and full doses of chloral and small doses of morphia were administered at bed-time. The following day he was better, but two days later he had a relapse. The house surgeons, Dr. Montegut and Dr. Marcus, repeated the leeching and increased the number of instillations of the eserine and cocaine solution. In

addition to the hypnotics at night, mercurial inunctions were ordered, two drachms being rubbed into the skin of the abdomen during each twenty-four hours. By the end of the month he was vastly improved and could readily tell the time of day on a watch. After this his recovery was uninterrupted. The eye became white and quiet, the cornea transparent, the coloboma only slightly obstructed by a few strands of capsule, and the vitreous perfectly clear. The disc is a vertical oval of good color, and no splotches are visible in the fundus. In February of the present year his vision, with + 12, spherical, is $\frac{6}{12}$ and part of $\frac{6}{9}$, and with + 16ª he reads Jäger I, quite readily.

Remarks.—This case seems worthy of record on account of the somewhat remarkable series of diseases, as well as surgical events, experienced by this patient. The gummatous irido-cyclitis was followed, as an almost necessary consequence, by secondary cataract, and the extraction of this without accident was of itself a gratifying feature. The development of a series of symptoms, which may with propriety be designated as glaucoma after extraction, is not so readily explained. There is, however, a slight entanglement of the iris in the upper and inner part of the operation scar, and it is quite possible the use of atropine for one day, when the symptoms first developed, was a piece of injudicious therapeutics, which determined the rapid rise of tension within a few hours and the violent pain which followed. The speedy relief of the condition under the influence of cocaine and eserine, aided by copious blood-letting, is interesting, and I cannot help thinking that the full doses of chloral, together with a rapid mercurialization, were efficient aids in the happy result. (See No. 19, Table of Cases.)

CASE III.—*Nuclear Cataract; Artificial Ripening by Direct Trituration; Extraction followed by Prolonged and at times Violent Dementia; Recovery of Reason and Good Vision.*[1]

Although this case, like the preceding one, has been reported, it naturally belongs to the series here recorded, and is therefore reproduced, so far as its most important features are concerned.

Diller Shirk, aged fifty-nine years, a patient in the Ophthalmic Wards of the Philadelphia Hospital, had been suffering with cataract for about twenty months, in each eye, nuclear in character, very slow in development, and most marked upon the left side. In addition to his visual defects, he had organic heart disease, with loud, double murmurs over the aortic cartilege, and almost constant, and at times serious cardiac vertigo. The urine contained albumin and casts. He was greatly depressed by his general condition, but especially by his inability to read.

As the growth of the cataract was so slow, on November 14, 1892, a preliminary iridectomy was performed and the capsule of the lens triturated with a small horn spatula. Advance of the cataractous process began at once, and by December 16th the lens was of a thick white color in its entirety. On the 30th, it was extracted through a 3 mm. flap, resulting in a perfectly black pupil and the ready counting of fingers.

For two days the patient was in perfectly good condition, but on the night of

[1] This case has been reported in full in *Annals of Ophthalmology and Otology*, Vol. II, 1893, p. 143.

the third day after the operation he became delirious, got out of bed, struck his eye, burst open the wound, causing a blood-clot which half-filled the anterior chamber. He now passed into a state of dementia, often of a violent character, necessitating constant watching. Emaciation became marked; there was involuntary passage of urine and fæces; liquid food was administered with difficulty; the pulse was exceedingly feeble and irregular; some hypostatic congestion of both bases of his lungs became evident, and thick sordes formed on the teeth and tongue.

The treatment consisted of free stimulation with whiskey, condensed and for the most part liquid nourishment, and full doses of strychnia and digitalis. The drugs, however, appeared to have no influence upon the circulation, and consequently the resident surgeon, Dr. Montegut, began the administration of nitro-glycerine, pushing it to its full physiological effect, Very soon improvement began, and by the last week in January he had recovered sufficiently to sit up for a few hours of the time, although he was still somewhat demented and required watching. This improvement has continued, and at the present writing, February 20, 1893, his mental derangement has largely disappeared, he eats solid food with avidity, his nutrition has improved, and in all respects he has practically recovered. Curiously enough, during the whole of this time there was no unfavorable symptom in the eye. The blood-clot caused by the blow was speedily absorbed, and save some slight conjunctival redness, which lasted for a few days, there was no reaction. The coloboma is clear, the eye-ground healthy, and the vision with his glasses equals 20/XXX, and he reads Jäger I. (See No. 20, Table of Cases.)

Remarks.—The subject of mania after cataract extraction has so recently been the subject of extended studies by Fromaget, Posey, and others that it does not seem necessary to enter into a discussion of the subject here. Suffice it to say, that it is evident that no one cause can explain all the cases. Perhaps this insanity is most apt to occur in those individuals whose mental balance is not entirely secure before operation, as apparently was the case with the patient described. Doubtless, many of the cases occur from a species of auto-infection, as has been insisted upon by Fromaget and others, while it would seem very sure that bandaging is sometimes responsible for the insanity. In curious confirmation of this fact it may be stated that the right eye of Diller Shirk was submitted to operation just one year after the operation upon the left eye, and, at his request, the non-operated eye—that is, the one which had secured sight by the previous operation—was allowed to remain unbandaged. Convalescence was uninterrupted and there was not the slightest sign of dementia.

CASES IV, V, VI, VII, VIII.—*Five Cases of High Myopia; Extraction of the Partially Opaque Lenses with Good Results in All.*

Mary Sullivan, aged seventy-five years, was admitted to the Philadelphia Hospital with double cataracts and high myopia. The right lens was extracted by

the combined method on June 4, 1894, and the healing was uneventful except that on the third day the wound reopened, but speedily reclosed. From the left eye the lens was extracted after preliminary iridectomy, both lenses being lifted with the loupe. At neither operation was there an escape of vitreous. After healing, the ophthalmoscope showed a huge posterior staphyloma, everywhere extensive myopic, atrophic retino-choroiditis, the splotches being so close together that it was difficult to realize how the patient obtained the vision which she did, namely, without a glass, 5/40. (See Nos. 35 and 52, Table of Cases.)

Kate Shevelin, aged forty years, was admitted to the Philadelphia Hospital with immature nuclear cataracts, myopia and lachrymal obstruction. After preliminary iridectomy, the lenses were extracted, the recovery uneventful, the vision in each instance being 6/30 with + 1ˢ. No attempt was made to correct the astigmatism if it existed. The ophthalmoscope revealed in each eye numerous vitreous opacities, huge posterior staphylomas and extensive myopic choroiditis. (See Nos. 42 and 45, Table of Cases.)

Mary C., aged seventy-five years, somewhat demented, was admitted to the Philadelphia Hospital, June 7, 1897, with double cataracts, that upon the right side being over-ripe, and the nucleus dislocated downward. From the shape of the ball and the history of the case myopia was suspected. The light projection was imperfect. An ordinary 3 mm. incision was made, with iridectomy, the capsule proving utterly resisting to the point of the cystotome. The lens was expelled in the capsule by external manipulation, and was followed by a smart gush of vitreous. The cataract thus removed can best be described by saying that it resembled a minute gum ball, and felt like a tissue of this character to the touch. As the flap was in place, the lids were closed and the bandage not removed for four days, when the wound was found healed. On the seventh day, the patient's eyes were affected with acute contagious conjunctivitis, then somewhat prevalent in the city and very prevalent in certain portions of the hospital. Recovery was perfectly good, although convalescence was prolonged. The vision without glasses was 4/40, although the exact vision was difficult to obtain on account of patient's mental condition. (See No. 55, Table of Cases.)

Remarks.—The chief interest that attaches to the first case, is the promptness of recovery after extraction in spite of discouraging circumstances, and the very good vision which was obtained although the fundus lesions were so extensive. In two of the cases, namely, Mary Sullivan and Kate Shevelin, the myopia could not have been less than twelve dioptres prior to the extraction, and probably it is higher. It was difficult to determine in the last patient what the myopia was, or, indeed, difficult to make anything like an accurate examination, owing to her mental condition. It is surprising that she recovered with anything like useful vision, when one considers the large loss of vitreous and the attack of conjunctivitis which began on the seventh day. As nearly as could be told, her vision is that recorded, namely, 4/40 without a glass. She disappeared soon after-

ward, and has not been seen since; therefore, whether this vision continued cannot be stated.

CASE IX.—*Chronic Granular Lids; Combined Extraction of Cataract; Normal Healing.*

Margaret Grundy, aged sixty years, was admitted to the Philadelphia Hospital with partial cataract upon the right side, and fully-developed cataract upon the left, and the early stage of cicatricial trachoma; that is to say, white scar lines were beginning to appear among the remains of old granulations; little or no discharge and no pannus; at most, a little haziness of the corneal epithelium. After thorough irrigation of the conjunctival cul-de-sac for several days with bichloride of mercury, 1 : 8000 (a lotion which I used in those days), an ordinary combined extraction was performed, the incision lying in the corneo-scleral margin, and the flap 4 mm. in height, the eye lightly bandaged after twenty-four hours. At the end of that time the bandage was removed and the patient given no protection except dark glasses. The conjunctiva was frequently irrigated with the aforesaid antiseptic lotion. Healing was uncomplicated, and with + 11, the vision was 20/70 without discission. The patient remained around the hospital for some months, then disappeared, and then reappeared at the end of two years, being admitted for acute exacerbation of the trachoma, and well-marked beginning pannus in the cornea of the eye on which I had operated. After treatment there was sufficient subsidence of the trachomatous process again to permit of sufficient vision to allow her to walk about. The haziness which had developed over the coloboma, however, destroyed reading vision. (See No. 1, Table of Cases.)

A precisely similar result was obtained in a second patient; Sarah ——, admitted to the Philadelphia Hospital, in which there was similar chronic trachoma, without pannus, the result being 20/70 after the extraction had healed. This patient was not again seen after leaving the hospital.

Comment.—These two cases, and several others which I have seen, confirm a belief, which I have long held, that granular lids at that stage are not likely to infect the wound, but that for fear discharge should accumulate beneath the closed lids, bandaging should be dispensed with as early as possible, and the conjunctival sac frequently cleansed after operation. (See No. 2, Table of Cases.)

CASE X.—*Clonic Blepharospasm; Lateral Nystagmus; Simple Extraction; Normal Healing.*

Mary Gallagher, aged sixty-six years, was admitted to the Philadelphia Hospital with double cataracts, mature on the left side. The patient had Pott's disease, which had existed since childhood; there were albumin and tube-casts in the urine, and almost constant blinking movements of the eyelids, a form of clonic blepharospasm, with slightly thickened conjunctiva, and moderate conjunctival discharge gathered at the commissural angles. There was rapid lateral nystagmus, and from the patient's statements it would seem that she had always been myopic. After

treating the conjunctiva for some days, the left cataract was extracted through Knapp's section, the blepharospasm being somewhat controlled by several instillations of cocaine, the nystagmus, of course, unaffected. The lens was delivered without difficulty and the speculum removed, when an unusual attack of blepharospasm appeared, causing quite a smart escape of vitreous and temporary eversion of the flap. This was immediately replaced, a slightly compressing bandage applied and the patient put to bed. There was absolutely no complication in the healing. The pupil was circular and the media clear, showing a large posterior staphyloma with spots of myopic choroiditis throughout the eye-ground. Vision with $+ 9 \supset + 2.75^{c}$, axis 180, 6/27. The right cataract was subsequently extracted without complication. (See Nos. 18 and 77, Table of Cases.)

CASE XI.—*Clonic Blepharospasm with Chronic Conjunctivitis; Combined Extraction; Loss of Vitreous; Normal Healing.*

John Fry, aged seventy years, was admitted to the Philadelphia Hospital, January 19, 1898, with ripe left cataract. On the right side the cataract had been extracted elsewhere, but the visual results were negative, probably due to detachment of the retina. No view of the fundus was obtained. The left cataract was extracted by the combined method through a 3 mm. flap, but the patient's blepharospasm apparently was aggravated by the operative interference, and in spite of every precaution, there was sharp squeezing of the lids down upon the eyeball, which was somewhat prominent, and a smart gush of vitreous. The lids were immediately closed, and a pressure bandage applied, no attempt being made to examine the condition of the wound, lest a renewed attack of blepharospasm would completely evacuate the eyeball. The bandage was not removed for five days, at the end of which time the wound was found perfectly healed, the pillars of the coloboma in place, and the vision good. About two months later, with $+ 12 \supset + 3^{c}$, axis 180, the vision was 5/15. No discission was attempted. The patient has been seen once since, about six months after the extraction, the vision being quite as good then as that recorded. (See No. 63, Table of Cases.)

CASE XII.—*Sub-conjunctival Dislocation of the Lens; Removal and Restoration of Vision.*

Edward Christopher, aged forty-six, was admitted to the Philadelphia Hospital, on December 10, 1900, with the following history: In June, 1900, he was struck in the right eye during a fight, probably with a set of false knuckles. The eye was ruptured, hopelessly lost, and enucleated at the Medico-Chirurgical Hospital. Up to that period his left eye had been fairly serviceable, although he had been told years before that he was getting a cataract. After convalescence he continued his work, that of a day laborer, until November 30th, without special inconvenience. On that day he fell, probably while intoxicated, and struck his right eye against a telegraph pole or the curb, he does not know which. He was taken home and received no medical treatment until he was brought to the Philadelphia Hospital, ten days later.

Examination showed on the right side an empty socket and a good stump. On the left side were the following conditions: The cornea was slightly hazy, the anterior chamber and the anterior layers of the vitreous partially filled with

streaked blood-clot. The iris could be distinguished downward and outward, but upward and inward was wanting, as it would be after a large iridectomy had been performed. Beginning at the upper and inner corneo-scleral margin there was a large swelling, somewhat resembling a huge corneo-scleral staphyloma covered with translucent conjunctiva through which passed coarsely injected blood-vessels.

The base of the swelling was stained bluish-black with uveal pigment. The diagnosis of subconjunctival dislocation of the lens was easily made. The apex of the swelling was incised with a cataract knife, and immediately the entirely opaque crystalline lens escaped. The sac in which it lay was filled with grumous material, doubtless the remains of the softened cortex. Vision immediately preceding and following the operation was shadows, or perhaps uncertain counting of fingers held close to the eye. The usual cataract dressing was applied, and the patient placed first upon small doses of mercury and later upon iodide of sodium. Healing was uneventful, and the eye soon became white and quiet, presenting exactly the appearances of one upon which the operation of combined extraction had been performed, with an unusually large iridectomy. Seven weeks later, with a $+3 \supset +6^{\circ}$ axis H, the patient could read 5/15 and most of 5/10. The fundus could be seen somewhat indistinctly, owing to the fact that the vitreous was still slightly cloudy, and in its anterior layers were several large blood-stained masses, probably clots, not yet absorbed. This case does not strictly belong in the list of cataract operations, but is here recorded as an interesting example of severe traumatism with an unusually happy ending, the vision being quite as good as after many normal cataract extractions.

TABLE OF CASES.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
1	Margaret Grundy.	60	F.	Good.	Chronic granular lids.	Mature senile, O. S.	1, 20, '88.	Combined extraction, incision in corneo-scleral junction, 4 mm. from upper tangent.	Uneventful.	20/LXX.		+ 11.s, 20/L.	Patient seen about two years afterward, and had in the meantime had an attack of acute granular lids with pannus on cornea; vision, however, still sufficient to enable her to get about.
2	Sarah.	60	F.	Good.	Chronic granular lids.	Mature senile, O. S.	1, 20, '88.	Combined extraction, 4 mm. flap.	Uneventful.	20/LXX.		+11.s 20/LXX.	Patient had chronic granular lids, but no active manifestation of disease at time of operation.
3	Charles Lewis. (Colored.)	41	M.	Feeble; constitutional syphilis.	Secondary glaucoma.	Complicated secondary, O. S.	1, —, '89.	Iridectomy one year before; extraction through corneo-scleral incision.	Uneventful.	Counts fingers, lower outer field.		Counts fingers, lower outer field.	Deeply-cupped atrophic nerve, the result of glaucoma following iritis.
4	Ellen Coles.	48	F.	Good.	High myopia; luxated lens	Complete, luxated into anterior chamber, O. S.	2, —, '89.	Lens extracted through incision in lower corneo-scleral junction.	Uneventful.	10/cc.		10/c without glass.	Extensive myopic choroiditis; myopia neutralized by extraction of lens. No glass required for distance.
5	Peter McDonald.	49	M.	Good.	Good.	Mature senile, O. S.	7, 12, '89.	Combined extraction, 3 mm. flap.	Uneventful.			+ 10 ⊃ + 75° ax. 150, 20/XX.	
6	Italian male.	70	M.	Organic heart disease.	Good.	Mature senile, O. S.	1, —, '90.	Simple extraction, Knapp's section.	Uneventful.	20/L.		20/L with + 14.	
7	James McMahon.	58	M.	Chronic alcoholic; albuminuria.	Good.	Immature senile, O. S.	1, —, '90.	Preliminary iridectomy, March, 1889; extraction, January 18, 1890; some cortex remains in coloboma.	Uneventful.	Counts fingers.	Three discissions.	Counts fingers.	Small atrophic disc; shrunken arteries. Patient able to get about. Clear coloboma.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
8	Eliza Murphy.	50	F.	Good.	Good.	Unilateral hypermature, O. D.	1, 25, '90.	Combined extraction, 3 mm. flap; nucleus dislocated into vitreous.	Uneventful.	Tells time on watch.		20/c with + 10.	When cystotome touched lens, field of operation flooded with milky fluid; sudden movement of patient dislocated nucleus into vitreous; three attempts with loupe failed to recover it; coloboma perfectly clear. Six months later, patient had good vision; small nucleus could be seen at bottom of vitreous; subsequently some operation, probably discission, at hands of another surgeon, resulted in iridocyclitis and ultimate loss of vision.
9	Peter McDonald.	49	M.	Good.	Good.	Mature senile, O. D.	10, —, '90.	Combined extraction.	Uneventful.	20/L.	Discission.	+ 11.ª 20/xx.	
10	Emmanuel Mack	75	M.	Good.	Former injury; large corneal scar; occluded pupil.	Shrunken traumatic, O. D.	12. 9 '90.	Shrivelled lens, removed through corneal incision combined with iridectomy.	Uneventful.			20/cc with + 18	There was a large corneal scar, the result of a knife-thrust, which had caused the traumatic cataract.
11	Sarah Ann.	89	F.	Senile dementia.	Good.	Mature senile, O. D.	12, 9, '90.	Combined extraction, 3 mm. flap.	Uneventful.			With + 11. 20/L.	
12	Emmanuel Mack.	75	M.	Good.	Corneal macula since childhood.	Complicated, following ulcers of cornea, O. S.	6, 12, '91.	Preliminary iridectomy, May 22, 1891; extraction 3 mm. flap, June 12, 1891. Some cortical remnants.	Uneventful.	10/cc.	Two discissions, Dec. 9, 1891, and Dec. 21, 1891. May 5, 1892, Agnew's capsule operation.	10/cc.	Large corneal macula, which had existed since childhood; huge posterior staphyloma and disseminated choroiditis.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
13	Sarah Ann.	89	F.	Senile dementia and bronchitis.	Conjunctival hyperæmia.	Mature senile, O. S.	6, 12, '91.	Combined extraction. Patient very unruly	Localized infection of flap and iritis.	Light perception.		Light perception.	Patient delirious and uncontrolled during convalescence, frequently pulling off bandages; kerato-iritis, followed by opacity of cornea. When last seen, there appeared to be still light perception, although the ball was soft; patient too insane to make positive demonstration.
14	Mrs. B. Hoffauer.	60	F.	Good.	Good.	Mature senile, O. S.	6, 12, '91.	Combined extraction.	Striped keratitis.	20/c.		+ 10 ⌒ − 3c ax. 15, 20/xx.	No fundus lesions; two years later had attack of kerato-iritis in that eye, and recovered with useful vision.
15	Patrick Mooney.	69	M.	Good.	Good.	Mature senile.	11, 16, '91.	Combined extraction.	Uneventful.	20/cc.	Discission, one month later.	+ 12 ⌒ 3c ax. 165, 20/lxx.	
16	Hugh Costello.	40	M.	Good.	Good.	Mature bluish-white, O. S.	12, 9, '91.	Combined extraction: lens removed in capsule.	Uneventful.	20/xxx.		+ 6 ⌒ + 1c ax. 90, 5/10, November 12, 1897, V. = 5/5.	The ultimate vision was recorded nearly four years after the primary operation; there were then small, floating, vitreous opacities and a typical myopic fundus, with myopic staphyloma.
17		49	M.	Good.	Good.	Mature senile.	5, 9, '92.	Knapp's section; simple extraction; one hour later small prolapse excised.	Uneventful.	6/60.	Discission, five weeks later.	+ 11 ⌒ + 3c ax. 75, 6/15.	
18	Mary Gallagher.	66	F.	Chronic Bright's disease.	Blepharospasm and lateral nystagmus.	Complicated senile, O. S.	7, 8, '92.	Simple extraction; Knapp's section; much squeezing; loss of vitreous.	Uneventful.	6/27.		20/lxx with + 9 ⌒ + 2.75c ax. H.	There was rapid lateral nystagmus, and the ophthalmoscope revealed large splotches of retino-choroiditis; posterior staphyloma; shallow cupping of disc.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
19	Robert Kingsbury.	50	M.	Tertiary syphilis.	Former gummatous iritis.	Complicated following iritis, O. D.	11, 28, '92.	Preliminary iridectomy, November 7, 1892; extraction, November 28, 1892,	Uneventful until fifteenth day, when an attack of glaucoma set in; cured by eserine.	6/12.		6/9 with + 12.	This very remarkable case has been published in full in *Annals of Ophthalmology and Otology*, Vol. II, No. 2.
20	Diller Shirk.	59	M.	Chronic heart disease.	Good.	Immature nuclear, O. S.	12, 30, '92.	Preliminary iridectomy and direct massage of lens, April 14, 1892; extraction 3 mm. flap, December 30, 1892.	Operation, followed by insanity, lasting three months.	6/30.	Discission.	6/9 with + 11.^{s}, November 15, 1897, V. = 5/15.	This interesting case of insanity following operation has been reported in *Annals of Ophthalmology and Otology*, Vol. II, No. 2.
21	Catherine Williamson.	78	F.	Feeble.	Chronic conjunctivitis.	Mature senile, O. D.	5, 3, '93.	Simple extraction followed by a small iridectomy an hour later on account of small prolapse.	Uneventful.	6/22.		6/22 + 12.	Although there was considerable web in pupil, discission, which would have raised vision, was declined.
22	Kate Gallagher.	60	F.	Insane.	Good.	Mature senile, O. D.	5, 11, 93.	Simple extraction.	Uneventful	6/12.		6/12 with + 12.	
23	Rosina Schmidt.	70	F.	Good.	Good.	Mature senile, O. D.	5, 22, '93.	Simple extraction; Knapp's section.	Prolapse of iris, on fourth day, followed by iritis.	Sees time on watch with + 12.	Declined.	Counts fingers.	Six months after extraction pupil was drawn upward and occluded by a web; patient could count fingers, but declined to allow iridectomy or other operation.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
24	John Duffy.	79	M.	Insane.	Moderate chronic conjunctivitis.	Mature senile, O. D.	5, 22, '93.	Simple extraction; patient violent and perfectly uncontrolled; large prolapse of vitreous, but lens safely delivered.	Uneventful.	Counts fingers.		Counts fingers.	Patient became violently insane during operation, striking at operator and squeezing up eyelids while knife was still in anterior chamber; in spite of this, there was no prolapse of iris and wound healed kindly with, however, a whitish mass in pupil; further operative interference was impossible—in fact, examination was practically out of the question, as patient remained in a demented state. One year later, eye was in much the same condition, although probably having no more vision than light perception.
25	Kate Gallagher.	60	F.	Insane; albuminuria.	Good.	Mature senile, O. S.	6, 14, '93.	Combined extraction, 3 mm. flap.	Sudden death at end of forty-eight hours.				Two days after operation patient died suddenly; autopsy forbidden; urine contained albumin; patient had counted fingers on previous day.
26	Agnes Donovan.	65	F.	Feeble.	Good.	Nuclear.	6, 26, '93.	Preliminary iridectomy and trituration of lens, May 17, 1893; extraction, June 26, 1893, 3 mm. flap.	Immediately following extraction, furious intraocular hemorrhage.			O.	This case has been fully reported in *Annals of Ophthalmology and Otology*, Vol. III. No. 1. Suppuration did not supervene, but the eye became quadrate, although the cornea remained clear but slightly ecstatic.
27	Catherine Williamson.	78	F.	Feeble.	Chronic conjunctivitis.	Mature senile, O. S.	11, 1, '93.	Combined extraction, 3 mm. flap.	Uneventful.	6/22.		6/22.	Some thickening of capsule, but secondary operation declined.
28	Rosina Schmidt.	70	F.	Good.	Good.	Mature senile, O. S.	11, 1, '93.	Combined extraction, 3 mm. flap.	Uneventful.	6/9.		6/9 with + 14.	Right eye had kerato-iritis after simple extraction. See No. 23.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
29	John Mackin.	52	M.	Good.	Good.	Mature senile, O. S.	11, 10, '93.	Preliminary iridectomy by another surgeon; extraction, November 10, 1893; some cortex remaining in pupil.	Uneventful.	6/15.	Discission.	6/6 14*.	
30	Diller Shirk.	60	M.	Chronic heart disease.	Good.	Imm ture nuclear, O. D.	12, 8, '93.	Combined extraction, 3 mm. flap.	Uneventful.	6/30.	Discission.	6/12 11.	Right eye of patient, who a year before had insanity following extraction of left cataract.
31	Samuel Collins. (Colored).	39	M.	Phthisis.	Large adherent leucoma.	Complicated following ulcer, O. S.	12, 26, '93.	Anterior synechia divided, Nov. 24, 1893; iridectomy, December 15, 1893; extraction through corneal incision of cataractous lens which partially filled anterior chamber.	Slow and complicated with iritis.	Light perception.		Light perception.	This patient had only faint light perception in lower and outer field, and no hopes of much improvement were held out to him. The vision after extraction was the same as that before it—namely, light perception in the lower and outer field. Three months later eye became irritable and was enucleated by another surgeon. History of case at this time unobtainable.
32	John Elliott.	40	M.	Feeble.	Doubtful light perception, except above; probable detachment of retina.	Complicated milky-white, O. D.	5, 9, '94.	Iridectomy five years before by another surgeon; extraction through 3 mm. flap; free escape of fluid vitreous.	Vitreous continued to ooze for five days, when wound closed; tension diminished.	Light perception, as before.		Light perception.	This was probably a case of extensive detachment of retina, and no hope of restoring vision was held out to patient, but he begged for extraction of lens; in spite of oozing of vitreous for five days, wound closed, the pupil being somewhat horizontally oval and drawn upward; a red reflex was obtainable, but no details of fundus; several months later patient was seen with eye slightly quadrate, but still with faint light perception in upper portion of field as before; has not been seen since.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
33	James Beiderman.	64	M.	Good.	Adherent corneal leucoma; iridectomy seven years before.	Chalky lens, O. D.	5, 9, '94.	Preliminary iridectomy seven years before; lens extracted through 3 mm. flap.	Uneventful.	5/40.		5/40 + 15.	The leucoma was a little below the centre, giving a clear pupil above, through which the patient obained the vision recorded. The leucoma had been the result of an ulcer many years before.
34	James Clayton.	26	M.	Inherited syphilis; ozena.	Former interstitial keratitis; central corneal macula.	Complicated, O. S.	6, 1,' 94.	Linear extraction; incision with broad keratome; no iridectomy.	Uneventful.	3/30.		+ 12. 3/30; reads large letters with + 20.	Considering the character of the cornea and frightful ozena, this may be considered as a somewhat remarkable result; no good view of fundus could be obtained on account of extensive vitreous opacities.
35	Mary Sullivan.	75	F.	Feeble; chronic rhinopharyngitis and catarrhal deafness.	High myopia; semi-luxated lens.	Complete, O. S.	6, 4, '94.	Combined extraction, 3 mm. flap; lens removed in capsules.	Uneventful.	Counts fingers.		Without glass, 5/40.	Ophthalmoscope shows huge posterior staphyloma, extensive myopic, atrophic retinochoroiditis everywhere. It is difficult to test the vision on account of the patient's mental condition, but apparently it is as recorded; the myopia has been neutralized by the extraction of the lens.
36	Nellie Pierce. (Colored.)	60	F.	Good.	Good.	Mature senile, O. S.	6, 9, '94.	Combined extraction; coloboma somewhat irregular and cortex remaining in pupil.	Uneventful.	5/40.	Discission.	5/10.	
37	Patrick Munay.	39	M.	Good.	Good.	Unilateral white, O. D.	11, 7, '94.	Simple extraction; Knapp's flap.	Uneventful.	6/30.	Discission.	+ 9 ⊂ + 1.50° ax. H 6/15.	The pupil perfectly mobile, slightly oval, and two posterior synechiæ
38	Rose McKeever.	60	F.	Dementia.	Good.	Mature senile, O. D.; greatly thickened capsule.	11, 19, '94.	Simple extraction; Knapp's section; dense capsule with calcareous changes.	Uneventful.	Counts fingers.	Two discissions, making only small hole in centre of capsule.	With + 10, 20/cc.	Patient's mental condition at this time did not permit a further operation on capsule, which would have best been treated by excision, as it was too dense for needling.

TABLE OF CASES—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
39	Rose McKeever.	60	F.	Dementia.	Good.	Mature senile, O. S.	12, 29, '94.	Combined extraction; much hemorrhage from iris.	Slight striped keratitis and incarceration of iris in angle of wound.	20/C.		20/C with + 10; reads large newspaper print.	
40	Thomas Maxwell.	50	M.	Good.	Good; unusual depth of orbit.	Mature senile, O. D.	1, 9, '95.	Combined extraction; short flap; unusual depth of orbit.	Uneventful, except for slow closure of wound owing to small tag of capsule caught in it.	Tells time on watch.		+ 11, 20/L.	
41	James Russell.	45	M.	Good.	Good.	Unilateral white, O. S.	1, 28, '95.	Simple extraction; Knapp's section.	Uneventful.	Tells time on watch.		+ 11, 6/6.	
42	Kate Shevelin.	40	F.	Feeble.	Lachrymal obstruction.	Immature nuclear, O. D.	5, 17, '95.	Preliminary iridectomy, December 14, 1894; extraction, May 17, 1895.	Uneventful; small prolapse of anterior pillar of coloboma.	5/20.		+ 1ª, 6/30.	Patient highly myopic; correcting lens + 1; many vitreous opacities; huge posterior staphyloma and myopic choroiditis.
43	Ann Burns.	70	F.	Good.	Tremulous iris; fluid vitreous.	Mature senile, O. D.	6 21, '95.	Simple extraction; Knapp's section; prolapse of vitreous.	Uneventful.	Counts fingers.		+ 10, 6/60.	Patient had a dislocated lens in the vitreous on the opposite side; on the side operated upon numerous vitreous opacities.
44	Henry Hill.	58	M.	Good.	Good.	Immature nuclear, O. S.	11, 11, '95.	Preliminary iridectomy, June 10, 1895; extraction of lens, November 11, 1895; much cortex remaining.	Uneventful.	5/40.	Discission.	5/10 + 9 ⌒ 4° ax. H.	Tension minus; capsule remaining, but by discission large, clear space through web.
45	Kate Shevelin.	40	F.	Feeble.	Lachrymal obstruction.	Immature nuclear, O. S.	11, 20, '95.	Preliminary iridectomy, June 3, 1895; extraction, November 20, 1895.	Uneventful.	5/20.		6/30 + 1ª.	High myopia; staphyloma and retino-choroiditis; able to read with + 4.50; band of capsule across coloboma.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
46	Henry Hill.	58	M.	Good.	Good.	Immature nuclear, O.D.	12, 2, '95.	Preliminary iridectomy. June 28, 1895; lens extracted, December 2, 1895.	Uneventful.	5/22.	Discission.	5/10 + 9 ⌒ 4° ax. H.	Two small bands of capsule remaining across pupil.
47	Hugh Costello.	44	M.	Good.	Corneal macula from burn below pupil.	Mature, white, O. D.	12, 2, '95.	Simple extraction, followed an hour later by small iridectomy on account of tendency to prolapse.	Uneventful.	+ 7, 5/10.		+ 6 ⌒ + 2° ax. 45, 5/5.	
48	Kate Ingram.	50	F.	Insane.	Good.	Mature senile, O. D.	12, 2, '95.	Simple extraction.	Uneventful.	5/20.		+ 12, 5/15.	
49	Kate Ingram.	50	F.	Insane.	Good.	Mature senile, O. S.	12, 9, '95.	Simple extraction; Knapp's section.	Prolapse of iris at the end of twenty-four hours.	2/XL.	Abscission of prolapse, January 6, 1896, and wound stitched.	+ 9 ⌒ + 6° ax. 180, 5/20.	The healing after the excision of the prolapse and stitching of the wound with silk sutures was perfectly kind, the resulting pupil being a vertical oval.
50	William Clark.	27	M.	Albuminuria.	Good.	Soft, O. S.	12, 16, '95.	Simple extraction; Knapp's section.	Prolapse of iris; restoration under eserine on sixth day.	5/15.	Discission, January 16, 1896.	+ 9 ⌒ + 2.50° ax. 180, 5/5.	One strand across pupil; fundus normal and easily seen.
51	Annie Mooney.	65	F.	Good.	Chronic conjunctivitis.	Mature Senile, O. S.	11, 13, '96.	Combined extraction.	On fourth day, after traumatism in the night burst open the wound, violent iridocyclitis set in.	Fingers at one foot.	Not yet performed.	+ 12, 4/CL.	The eye became perfectly white and quiet, the pupil being drawn upward and closed with a patch of lymph, and either a successful iridectomy or iridotomy will restore useful vision, which even now is quite sufficient for her to get around the building.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
52	Mary Sullivan.	76	F.	Feeble; chronic rhino-pharyngitis; catarrhal deafness.	High myopia; semi-luxated lens.	Complicated, O. D.	12, 2, '96.	Preliminary iridectomy; extraction of lens in capsule, December 2, 1896.	Uneventful.	Counts fingers.		5/45.	Huge posterior staphyloma and extensive atrophic retino-choroiditis. See Case 35, for left eye of same patient.
53	Jane Martin.	79	F.	Senile dementia; albuminuria.	Good.	Mature Senile, O. D.	12, 2, '96.	Simple extraction	Uneventful.	Counts fingers.		Good.	Patient was a senile dement, and it was impossible to test her vision with types; she had a perfectly clear pupil, and saw readily across the ward.
54	N. Roberts.	51	M.	Chronic asthma and phthisis.	Chronic conjunctival hyperæmia.	Mature Senile, O. S.	12, 28, '96.	Extraction, 3 mm. flap; section terminating too far forward in cornea; some cortex remained; preliminary iridectomy.	Prolapse of iris in angle of wound; moderate iritis.	5/20.	Discission.	+ 11, 5/15 + 11 ⌒ 2c ax. 45, 5/10.	Patient has some thickening of capsule.
55	Mary S.	75	F.	Chronic bronchitis and Bright's disease; dementia.	Chronic conjunctival hyperæmia; fluid vitreous.	Over-ripe (Morgagnian), O. D.	6, 9, '97.	Combined extraction, O. D.; prolapse of vitreous.	No reaction; conjunctivitis on seventh day.	4/40.		4/40.	No reaction, but on seventh day conjunctivitis, which continued for several weeks; no infection of wound; conjunctivitis then epidemic.
56	James McAdams.	62	M.	Chronic bronchitis.	Good.	Hypermature, O. S.	11, 15, '97.	Preliminary iridectomy; extraction in capsule in O. S.	Uneventful.	+ 11, 5/10.		+ 12, 5/5.	
57	Louisa Jackson (Colored).	65	F.	Good.	Good.	Hypermature, O. S.	12, 3, '97.	Simple extraction, O. S.	Uneventful.	5/45.		+ 10 ⌒ + 2c ax. 180, 5/15.	
58	Bridget Byram.	52	F.	Good.	Good.	Mature senile, O. D.	12, 3, '97.	Simple extraction, O. D.	Uneventful.	6/45.		+ 11, 6/15.	
59	Louisa Jackson (Colored).	65	F.	Good.	Good.	Hypermature, O. D.	12, 10, '97.	Simple extraction, O. D.; prolapse of vitreous.	Uneventful.	5/45		+ 11, 5/15.	Some cortex remaining.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
60	Bridget Byram.	52	F.	Good.	Good.	Mature senile, O. S.	1, 7, '98.	Combined extraction, O. S.	Traumatism, seventh day; hyphæma.	Fingers.		+ 20, sees watch. Later, + 9, 5/40.	Thickened capsule; requires discission. Patient left without operation.
61	Catherine Heller.	50	F.	Chronic rheumatoid arthritis; melancholia.	Chronic conjunctivitis.	Nuclear, O. D.	1, 7, '98.	Extraction, 3 mm. flap; cortex very tenacious.	Uneventful.	5/45.	Discission.	+ 10 ⌒ + 3° ax. 165, 5/15.	Some thickened capsule; a year later patient returned with much thickening of capsule. Irido-cystectomy restored useful vision; it was repeated one year later.
62	Charles White.	55	M.	Dementia.	Secondary glaucoma.	Traumatic, lens, semi-luxated, O.D.	1, 7, '98.	Extraction, 3 mm. flap; collapse of cornea, O. D.	Uneventful.	5/45.		+ 12 ⌒ + 4° ax. 180, 5/20.	Cataract caused by blow six months ago; at that time secondary glaucoma, and iridectomy was done. Disc looks atrophic.
63	John Frey.	70	M.	Chronic bronchitis.	Clonic blepharospasm and conjunctivitis; liquid vitreous.	Sclerotic lens, O. S.	1, 19, '98.	Combined extraction, O. S.; blepharospasm; large loss of vitreous.	Normal healing.	Counts fingers.		+ 12 ⌒ + 3° ax. 180, 5/15.	Some grayish membranes in coloboma.
64	Thomas Morrow.	50	M.	Melancholia.	Secondary glaucoma following iritis.	Complicated, O. D.	1, 19, '98.	Iridectomy, June, 1897; extraction of right lens.	Uneventful.	Sees watch face.	Iridotomy.	+ 10, 1/45.	Much thickened capsule in coloboma; complete obliteration of nasal field.
65	Edward Cassidy.	51	M.	Good.	Good.	Unilateral white, O. D.	5, 16, '98.	Combined extraction, O. D.; lens dislocated and removed with loupe.	Uneventful.	+ 10, 5/25.		+ 10, 5/25.	
66	L. Thomas.	25	M.	Good.	Secondary glaucoma following traumatism.	Traumatic, O. S.	2, 20, '99.	Linear extraction	Uneventful.	Fingers.	Discission.	+ 11, 5/5.	

TABLE OF CASES—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
67	John Rohr.	54	M.	Good.	High myopia; calcareous dislocated lens.	Calcareous lens, O. D.	5, 17, '99.	Preliminary iridectomy; extraction of dislocated lens with loupe one week later.	Uneventful.	Shadows.		Shadows.	Eye-ground was the seat of extensive atrophic retino-choroiditis with atrophy of the disc.
68	B. Curley.	54	M.	Good.	Sympathetic ophthalmitis.	Complicated, after sympathetic ophthalmia, O. D.	12, 6, '99. 12, 13, '99.	Critchett's.	Uneventful	Counts fingers.		+ 10 ⌒ + 4° ax. 60, 20/L.	This remarkable case of recovery from sympathetic cataract is reported in full in the *Ophthalmic Record*, February, 1900.
69	Julius Wolf.	79	M.	Chronic Bright's disease. Melancholia.	Chronic conjunctivitis; fluid vitreous.	Over-ripe sub-luxated, O. D.	12, 21, '99.	Combined extraction; nucleus slipped into vitreous.	Intra-ocular hemorrhage.	Fingers.		Light perception.	While delivering soft lens matter the nucleus escaped into the semi-fluid vitreous. Attempts to dislodge it were not successful; five hours later there was a sharp intra-ocular hemorrhage, which burst open the wound and expelled the nucleus; hemorrhage controlled and wound closed with stitches; eye healed without suppuration; patient died about six weeks later, from the beginning being melancholic.
70	Owen Dorsey.	60	M.	Chronic bronchitis.	Good.	Mature senile, O. S.	1, 10, '00.	Combined extraction.	Uneventful.	5/15.		+ 11 ⌒ + 2° ax. 180, 20/XXX.	
71	James Gibson.	58	M.	Good.	Good.	Unilateral, white, O. D.	1, 24, '00.	Combined extraction.	Uneventful.	5/40.		+ 10, 5/10.	
72	Mary Jane.	60	F.	Good.	Good.	Mature senile, O. S.	11, 4, '95.	Combined extraction.	Uneventful.	6/12.	Discission.	+ 12 ⌒ 1.50° ax. H, 6/6.	
73	S. Desango.	39	M.	Melancholia.	Former iritis; secondary cataract; probable detachment of retina.	Complicated, following iritis, O. D.	6, 15, '96.	Combined extraction.	Successive attacks of hyphæma.	l. p.	Two discissions.	Sn. CC. held close to eyes.	No useful vision could be expected in this case of complicated cataract and retinal detachment, but patient was able to get around after operation.

TABLE OF CASES.—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
74	N. Temaroff.	60	M.	Good.	Good.	Mature senile, O. D.	1, 11, '92.	Simple extraction.	Uneventful.			Good.	Patient illiterate, and no record of vision with types; saw perfectly.
75	Mary Jane.	65	F.	Indifferent; recent severe accident.	Good.	Mature senile, O. D.	4, 13, '00.	Combined extraction.	Slow closure of wound.	6/60.		6/20 + 11s ⌒ + 4° axis H.	The slow closure of the wound led to the development of keratitis, leaving a corneal opacity. Three months after extraction this patient had an attack of iritis, but recovered perfectly.
76	John Mackin.	52	M.	Good.	Good.	Shrunken lens, O. D.	12, 8, '93.	Preliminary iridectomy by another surgeon seven years before.	Uneventful.	6/22.	Discission.	+ 12s ⌒ + 4° ax. 180, 6/6.	
77	Mary G.	66	F.	Bright's disease.	Chronic conjunctivitis; lateral nystagmus.	Mature senile, O. D.	8, —, '92.	Simple extraction.	Uneventful.			+ 10, 20/c.	There was a patch of calcareous degeneration of the capsule of the lens. The record a little obscure, probably the right eye of Case 18.
78	John Kane.	58	M.	Good.	Good.	Mature senile, O. D.	1, 10, '00.	Simple extraction.	Uneventful.	15/cc.	Discission.	+ 9 ⌒ + 75° ax. 180, 5/10.	
79	John Kane.	58	M.	Good.	Good.	Mature senile, O. S.	10, 5, '00.	Simple extraction.	Uneventful.	6/60.	Discission.	+ 10 ⌒ 1.50° ax. H, 6/15.	
80	George Martin.	55	M.	Good.	Good.	Mature senile, O. S.	10, 31, '00.	Simple extraction.	Uneventful.			+ 10 ⌒ + 75° ax. H, 6/9.	
81	Andrew Stenger.	75	M.	Good.	Horse-shoe-shaped macula of cornea.	Complicated, with corneal macula, O. S.	11, 9, '00.	Extraction after preliminary iridectomy.	Uneventful.	Fingers.		+ 10, 2/30.	The macular cornea precluded the possibility of better vision. The sight given was very satisfactory to patient.

TABLE OF CASES—Continued.

No.	Name.	Age.	Sex.	General Health.	Ocular Health.	Cataract.	Date.	Operation.	Recovery.	Immediate Vision.	Secondary Operation.	Ultimate Vision.	Remarks.
82	John Peters.	50	M.	Good.	Chronic conjunctival hyperæmia.	Immature, O. D.	11, 28, '00.	Extraction after preliminary iridectomy.	Uneventful.	5/40.		+ 9 ⌒ + 2c axis 15, 6/25.	The capsule is moderately thick, and requires discission.
83	Patrick McCorley.	65	M.	Arterio-sclerosis.	High myopia.	Semi-luxated, swinging in pupil, O. D.	11, 28, '00.	Simple extraction.	Uneventful.			+ 10, 5/15.	The lens, of amber color, swung to and fro in the pupil space, as if it were attached at either end of its horizontal diameter. No vitreous lost in the removal.
84	Jacob Breisch.	72	M.	Dementia.	Complicated with myopia, probably.	Mature and adherent to upper edge of iris, O. D.	12, 31, '00.	Combined extraction; lens delivered with loupe.	Uneventful.			+ 10, 20/cc.	There is thickened capsule with some cortex remaining. Discission required. Difficult to test vision, but apparently that given is approximately correct.
85	Edward Christopher.	60	M.	Good.	Ruptured corneo-scleral margin.	Lens luxated beneath conjunctiva, O. D.	12, 14, '00.	Removed by incising conjunctiva.	Uneventful.	Counting fingers.		+ 3 ⌒ + 6c axis H, 5/10.	The eye had been ruptured by a blow, and the lens dislocated beneath the conjunctiva.
86	Paul Sorg.	60	M.	Feeble.	Good.	Mature senile.	10, 12, '00.	Simple extraction, O D.	Uneventful.			+ 10, 5/40.	

REPORT OF A SUCCESSFUL CASE OF EXTENSIVE BLEPHAROPLASTY FOR THE REMOVAL OF AN EPITHELIOMA.

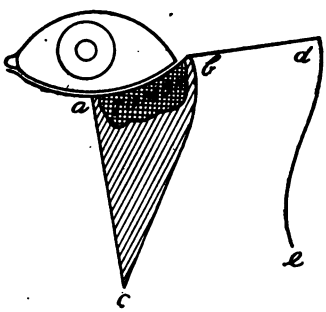
By CHARLES A. OLIVER, A.M., M.D.
OPHTHALMIC SURGEON TO THE HOSPITAL.

Through the kindness of Dr. B. J. Edger, J. G., a fifty-eight-year-old, man was admitted to my service at the hospital, on February 1, 1898, with the statement that for several years past he had noticed a small pustule upon the outer half of the left lower eyelid. The mass grew very slowly, and although it occasionally bled, yet it never gave him any pain.

Examination showed an epitheliomatous mass of about two centimetres' length and one centimetre in width going directly up to the ciliary border of the lid, but not encroaching upon the mucous surface, in the position above noted. It was broad based, immovably attached to the underlying tissues, bosselated, and presented many ulcerating points and areas of cystic degeneration upon its flattened surface. A slice of the tissue removed from the base of the mass proved the epitheliomatous nature of the tumor. The conjunctiva was uninvolved and the eyeball was healthy, unrestricted in movements, and performed all of its functions normally.

Two days later, while the patient was under the general anæsthetic influence of ether, and with the assistance of my resident surgeon, Dr. William Pepper, I performed the following operation for the removal of the growth, as can be better understood by the accompanying outline drawings. The mass with a surrounding rim of infiltrated healthy tissue was excised in the large triangular area *a b c* (Fig.

FIG. 1.

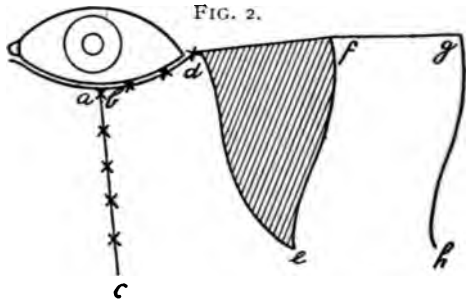


1), which included the external portion of the outer half of the lower tarsal border as a base (*a b*), with the two vertical sides *a c* and *b c*, terminating at the apex *c*. The medial border of the excised portion was dissected sufficiently loose to give it

a marked degree of pliability and freedom. In order to fill in this triangular gap, a quadrangular flap, *c b d e*, with its base, *b d*, equal in length to that (*a b*) of the denuded triangle and its two sides each equal in length, was made. During its formation, care was taken to make it as thin and as smooth as should be consistent with its health, its base being purposely broadened by an outward curve to the lower half of the side *d e*, in order to increase its chances of vascularity.

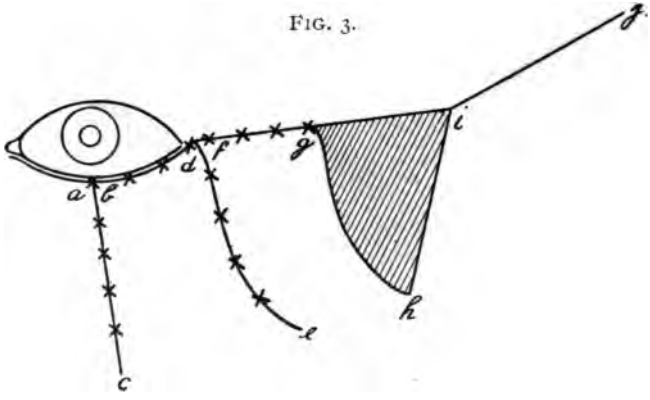
This done, all of the underlying tissues were freed from fat, thoroughly irrigated with warm sterile water, and the flap was slid over into position so as to cover the denuded area, and stitched with fine obliquely-placed superficial sutures in order that there might not be any puckering along the line of stitches, thus bringing *a* to *b*, and moving the line *d e* (as shown in Fig. 2) away from its temporal

FIG. 2.



border. The new ciliary border of the outer half of the lower lid, *b d*, was carefully stitched into position, thus fixing the flap, *c b d e*, into situation. In order to fill in the newly-exposed raw triangular area, *d e f*, a second flap, *e f g h*, similar in shape and size to the first one, was obtained in the same way, and set and similarly stitched into position, thus removing any cicatrizing area that might exert a harmful dragging influence upon the eyelids away from the vicinity of the eye. This left, as can be seen (Fig. 3), a third triangular area of denudation, *g h i*, which

FIG. 3.

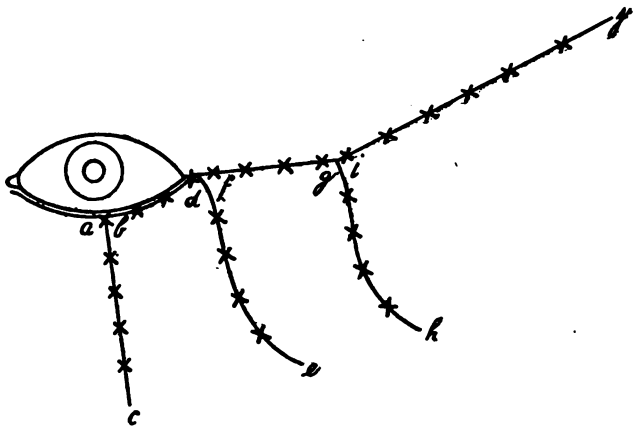


had to be covered by sound skin. To do this without leaving any nodules or irregular cicatrization lines, a long angular incision, *i j* (shown in Fig. 3), was made to a sufficient length, to give to the undermined triangular flap, *h i j*, easy

adaptation to be slid over and cover the area, *g h i*, the point, *i*, as shown in Fig. 4, being attached to the point *g*, and all of the free borders stretched into accurate approximation by obliquely-placed superficial sutures.

During the procedure all of the flaps were kept warm by heated sterile water, and every vestige of underlying blood-clots was removed before the flaps were set in place and sutured into position. The field of operation was cleansed and

FIG. 4.



thoroughly dusted with iodoform, and carefully covered with antiseptic gauze and a series of roller bandages.

In forty-eight hours' time the dressings were taken off, and several of the least important sutures were removed. Throughout the entire after-treatment, which was carefully watched over by Dr. Pepper, with most competent nursing, not a vestige of tumefaction or suppuration appeared; in fact, the case was practically cured in a week's time.

Two months later, the linear scars had become almost imperceptible.

Remarks.—The case is of interest not only on account of the thorough removal of the growth without the appearance of any remaining scar, but is of value in showing the expedients adopted to carry the possibility of any cicatrizing area away from the eyelids, and to prevent any granulation surface.

THE DIAGNOSTIC VALUE OF OCULAR CHANGES IN TUMOR OF THE CEREBELLUM.

By CHARLES A. OLIVER, A.M., M.D.,
OPHTHALMIC SURGEON TO THE HOSPITAL.

This case, with its characteristic general symptomatology and its post-mortem proving, is interesting from its ophthalmic standpoint alone; and for this reason has been given in its every detail as illustrative of the value of a thorough study of the eye-symptoms in all cases of suspected cerebellar growth.

The two optic nerves losing their conduction properties, gave rise to blindness. To these losses of sensory action, due to lymph stasis with consequent pressure-symptoms, as was shown at the post-mortem examination, by the blocking of the ventricular spaces and the undue tentorial tension, must be added the symptomatic want of light reflex with the preservation of the iris responses during associated muscular contracture—symptoms which, taken together, are highly designative of many of the cases of neoplasms situated in the cerebellar region.

The peculiar form of the ophthalmoscopic picture of neuro-retinal change is also highly characteristic of growth in this position. Not very pronounced, though dense, compact and squeezed dry, as it were, the nerve-head swelling often becomes representative of the disorder; far different from the purplish succulent types that are so frequently found with the cerebral form of lesion.

These two series of motor and sensory changes in the visual apparatus seen in all manner of relative degrees and intensities, are here offered from an ophthalmic standpoint, as an additional grouping of specialized symptoms that in themselves alone may be ofttimes considered of sufficient diagnostic import to give a solution to the not infrequent problem of the presence of cerebellar neoplasm.

The general history of the case in full is as follows:[1] In February, 1898, I was asked by Dr. James Hendrie Lloyd to see a case of supposed cerebellar tumor in the nervous wards of the hospital.

[1] For the original of these most carefully taken notes I am under obligation to my friend and former resident physician at the hospital, Dr. T. Percival Gerson, of Lansdowne, Pa.

The patient, F. E., an only child, was born in Philadelphia in March, 1877. The labor was prolonged and difficult, but no instruments were used.

At ten months of age the patient was breastfed by a wet-nurse, when almost immediately afterwards, in accordance with his mother's statement, small pustules appeared over the entire body. Five months later he had "spasms" that were supposed to be dependent upon dentition.

As a child, he had no other diseases but slight attacks of rubeola and pertussis.

In 1893, he was admitted to one of the State schoolships, upon which he made one cruise of six months' duration, being probably discharged for insubordination. Half a year after his return he contracted a gonorrhœal urethritis, which was treated at one of the large hospitals in this city. He was never known to masturbate. About this time he began to make free use of various alcoholic liquors. For a number of years he was lazy, kept late hours, and lived among evil associations.

There was never any history of traumatism. In August, 1897, he began to complain of frontal and occipital headaches, and more or less constantly held his head between his hands in the attitude of leaning over. Attacks of melancholia appeared. His eyes became expressionless and his hearing was impaired.

Four months later, it was noticed that his sight was gradually failing and that his hearing had grown worse.

Smell and taste never seemed to be affected. There was never any paresis or paralysis.

Appetite continued good until two weeks preceding his admission into the hospital. Before his present illness he was considered as always doing well in his school work. In November, 1897, it was noticed that he walked with his head hanging forward on his breast and that his eyes were kept rotated upwardly. He was unable—most probably in part due to his impaired hearing—to answer questions. He did not exhibit any illusions or delusions. He was never known to have any attacks of vertigo or exhibit any convulsive seizures.

Since January, 1898, he seemed to have developed an apathy for talking, although occasionally he would blurt out some of his wants. His mother stated that he had been sleeping a great deal, both by day and by night.

His father, who was an alcoholic, died of an injury. His mother was living and well. There was not any history of mental disorder or tuberculosis.

Careful physical examination, made on February 25th, showed the following conditions. Decubitus was on one side with the knees flexed and the thighs drawn up against the abdomen. The head was bent forward and the hands were clasped behind the neck. The skin of the abdomen was dry and harsh, with a few scattered, depressed, whitish scars. The body was spare and the legs and arms were somewhat wasted. The patient was more or less restless at times, but usually rested quietly. He would not respond when spoken to, seeming not to hear, even when the voice was forcibly transmitted directly into the ear; although he would often plaintively cry out for something, as "I want a cigarette," or give vent to the most passionate impulse, as "Fell him with a belaying-pin, mate!"

His hair was disheveled and sandy in tint. At times, while either lying quietly or complaining that his head ached most intensely, he would move his hand through his hair, clasping either the forehead or the occiput. There were no marks of cuts or wounds on the scalp.

The chest expansion was quite symmetrical. The right border of the sternum at the junctures of the costal cartilages was both prominent and knobbed. The

right lower chest anteriorly over the lower ribs was somewhat more hollow than the corresponding area upon the opposite side. The intercostal spaces could be readily palpated, while the supra-sternal notch and the supra-clavicular and infra-clavicular fossa were not difficult to see. The costal angle was about eighty degrees. The skin of the chest, like that of the abdomen, was dry and harsh, though the superficial veins were not unduly prominent.

Pneumonic palpation was uncertain, as the patient could not be made to speak. Percussion revealed a probable more resonance on the left side anteriorly than elsewhere. Posteriorly, upon the right side, there seemed to be an area of flatness which was continuous with that of the normal liver and extended beyond it. A similar area of flatness was found deeply situated in the corresponding axillary region.

Auscultation showed that the breathing sounds were seemingly normal anteriorly. Laterally, on the right side and above the liver posteriorly, an area of almost absolute negation of the breath-sounds was obtainable. No râles could be distinguished.

Liver dullness extended from the sixth intercostal space to one-half an inch above the costal margin in the mid-clavicular line ; while in the mid-axillary line it was continuous from the sixth to the tenth ribs.

No splenic dullness could be gotten.

The apex-beat of the heart was neither visible nor palpable. The sounds in the aortic region were quite pronounced, but no murmur could be detected. The sounds in the mitral region, which were more difficult to determine, gave evidence of an appreciable murmur.

The abdomen was somewhat scaphoid in shape with undue prominence of the processes. No points of tenderness could be elicited. Examination of the penis and scrotum showed the presence of a number of white blotches on the skin of the prepuce that contrasted quite markedly with the surrounding darker skin of the parts.

On the right hip, near the acetabulum, there was an abraded surface resembling a glazed-over bedsore, which was almost as large as a silver quarter of a dollar. Sensations to pin-pricks seemed at times to be delayed as long as four or five seconds. There was no demonstrable palsy in any part of the body, the patient moving his arms and limbs at will. The plantar reflex was slightly sluggish. The patellar tendon reflexes were almost absent. No patellar-clonus or toe-jerk could be obtained. Left ankle-clonus was lost.

Sensation about the head and face was much more acute than it was in the extremities. The mid-dorsal surface of the tongue was coated with a yellowish-white fur. The breath was foul.

The patient was apparently able to move his eyelids at will, but he did not wince when an object was rapidly moved before the eyes. He had a spasmodic cough, particularly during ingestion of food, he not having thorough control over deglutition. He drank all of the milk that was given to him, but he ate very little food that required mastication.

In attempting to get the patient out of bed, he was found to be too weak to even stand alone, there being a decided tendency to pitch both forward and laterally. During the night he had fallen out of bed, but did not suffer any injurious consequences. He had incontinence of both urine and fæces, though the bowels were somewhat constipated.

On February 24th (the day before examination), I made my first ophthalmic study.

The pupil of the right eye was dilated *ad maximum*. The iris was irresponsive to light stimulus, though the pupil varied considerably in size and in direct accordance with what it should, during the different conjoined movements of the two eyeballs.

The media of the eye were clear. There was a rather marked choking of the optic disc, with a pronounced haze and œdema of the circumjacent retina, though the prominence of the nerve-head was not very great.

The swelling of the nerve-head was dense and compact, and the swollen tissue did not seem to possess any degree of capillarity. The retinal arteries were reduced to threads, and the corresponding veins were unduly small. There were not any signs of recent or old hemorrhagic extravasations. There was a low degree of farsightedness with a minor amount of astigmatism.

The pupil of the left eye was the same size as that of the right eye. The iris behaved similarly with that of its fellow.

The media of the eye were transparent. The optic nerve-head was swollen to about the same degree as that of the right eye. The type of the neuro-retinitis was identical with that of its fellow. There was a want of capillarity in the substance of the disc. The retinal arteries, like those of the other eye, were greatly reduced in size and the related veins were much diminished. No signs of any hemorrhages or gross inflammatory products could be found.

Vision in each eye seemed to be lost.

The eyeballs possessed free and undisturbed motion, though at times a series of nystagmic impulses of brief excursive movement seemed to be present.

On the following day, the patient had a brief attack of vomiting, which is described as projectile in character, the ejecta being forcibly emitted some distance in front of the subject.

On the morning of February 27th, it was noticed that a small quantity of blood was oozing from his left nostril. Scattered over the face and the chest there were a number of papules.

The patient still complained when spoken to, asking, "Well, what do you want?" or suddenly exclaiming, "I'm awake!"

He was so weak as to be unable to stand, at times having a marked inclination to pitch forward. A slight degree of ankle-clonus on each side had become noticeable.

At times, he complained very greatly of headache, particularly if I attempted to direct the light of the ophthalmoscopic mirror upon the retina of either eye.

Examination of the auditory apparatus by one of my colleagues, Dr. John Morley Marshall, some two weeks after my first ophthalmic examination, failed to show any evidence of aural complication.

On March 23d, the case record reads: "From day to day the knee-jerk and the ankle-clonus have been elicited, with the result that a decided difference in their intensities have been noticed during each examination, they sometimes being increased, and at other times being diminished."

The choking sensation during the act of swallowing, dependent upon faulty deglutition, continued. The patient remained stupid and listless, though the cephalalgia did not appear to be so intense.

A few days later, it was noticed that his audition, like his vision, had disappeared (a tin can vigorously pounded near his ears not producing the slightest apparent auditory impression).

The incontinence of urine and fæces continued. He began to grow much weaker, and his skin assumed a much more anæmic appearance.

His treatment had been most varied, embracing alteratives, stimulants, digestives, and the most carefully regulated diet.

On March 3d of the same year, he painlessly sank, became comatose, and died. The necropsy notes bore out the ante-mortem diagnosis of tumor of the cerebellum. The autopsy was made by Doctors Lloyd and Bowman.

The body was that of a tall, much emaciated male, having several bedsores situated over the sacrum. There was a tattooing mark upon the flexor surface of the left forearm.

On opening the abdominal cavity the walls were noted to be thin and muscular and of dark color. The intestines were contracted. There was the usual amount of peritoneal fluid present. The appendix vermiformis was very long, its distal end being curled upon itself. The sigmoid flexure was elongated and distended with gas.

The pleura did not show any adhesions. The diaphragm extended to the fourth interspace on both sides. The pericardium, which contained about four fluid ounces of a yellowish fluid, was apparently normal.

The right auricle was distended with a chicken-fat and currant-jelly-like clot extending into the great vessels. The mitral orifice of the organ admitted three fingers. The valves were normal. The right ventricle was distended with clotted blood. The left auricle contained some clotted blood. The left ventricle was empty. The tricuspid orifice admitted two fingers. The valves were slightly thickened, showing areas of reddening. There were not any vegetations along the margin of the valves. The arch of the aorta appeared normal. The heart muscle was pale and flabby, the wall of the left ventricle being about one and a quarter centimetres thick.

The left lung was crepitant, the lower portion of the lower lobe being congested

and slightly œdematous. The external surface of lower lobe of the right lung presented an area of five centimetres in diameter. It was dark red in color, and at its centre there was a depressed suppurating area one and a quarter centimetres deep. The lung was crepitant throughout. Beneath the suppurating area there were other similar small ones.

The spleen and both supra-renal capsules were normal.

The left kidney was large. Its capsule stripped fairly well. It was dark red in color and its cortex was slightly narrowed. There was a considerable amount of fat in its peivis. The external surface of the right kidney showed a small (white) calcareous nodule. This kidney was smaller than its fellow; otherwise it was the same.

The ureters and the bladder were normal.

The liver was slightly enlarged. It cut with slight resistance and was firm. The gall-bladder was normal.

Upon removal of the skull-cap, a very large tumor was found between the vermiform process of the cerebellum and the overlying cerebral hemisphere. On careful dissection, this growth was easily shelled out of its bed. It was found to have only the slightest connection with the brain substance. It was encapsulated. By pressure downward it had flattened and destroyed the appearance of the quadrigeminal bodies and the veins of the cerebellum, but the aqueduct of Silvius was pervious, and the anterior medullary velum was unbroken.

The tumor growth was nodular, especially on its anterior superior aspect; in which situation, one large nodule, the size of a grape, had made pressure into the left cerebral hemisphere. The growth, which was very vascular, was six centimetres wide in its greatest diameter, six and a half centimetres long in its antero-posterior diameter, and four and a half centimetres in its vertical diameter.

On section, it proved to be a sarcoma. Its most striking characteristic, next to its size, was its comparative freedom from connection with the surrounding brain substance. There was no infiltration of any of the neoplastic tissue in the neighborhood. The ventricles of the brain were much distended. There were very large pacchyonian bodies situated along the course of the longitudinal fissure, these being deeply imbedded in the vault of the cranium.

The weight of organs was: Heart, 250 grammes; left lung, 510 grammes; right lung, 780 grammes; spleen, 110 grammes; left kidney, 170 grammes; right kidney, 140 grammes; liver, 1570 grammes, and brain, 1380 grammes.

THE TREATMENT OF RINGWORM OF THE SCALP.

By HENRY W. STELWAGON, M.D., PH.D.

The hospital which escapes the misfortune of having to care for cases of ringworm of the scalp has much for which to be grateful. Many such cases occurring in private practice, among well-cared-for children of good physique and robust health are, more especially if the disease is at all recent, usually rapidly responsive to well-advised measures. Not so with hospital and institution cases. Even in private practice observation shows that in many cases the disease is frequently rebellious and peristent, and that not infrequently the practitioner pronounces the cases cured when the hair has begun to fill in in the affected area or areas, whereas in reality the disease may still persist in a less conspicuous but chronic state, and the case remain an active centre of contagion for other children. In this hospital, as in fact in all institutions in which children with this disease are admitted, the subjects are almost invariably of poor stock, of impoverished nutrition, and have had, as a rule, the affection many months before coming under treatment. The consequence is, one has to deal with a persistent, rebellious and troublesome type of disease, scarce known to the average practitioner, but an all too common experience with the specialist in skin diseases. With persevering treatment even the most of these chronic cases may be made to yield in some months, or say within a period varying from several months to a year, depending somewhat upon the extent of the disease; occasionally a longer time is required for cases of great extent and of long duration. Without the methodical and persistent carrying-out of treatment, however, such cases last almost indefinitely—or till nature begins to look after the cure as the child verges into puberty, it disappearing spontaneously approaching or shortly after this age.

Ringworm of the scalp is in the larger number of cases due to the small-spored fungus, and, upon the whole, it may be accepted as a fact that this fungus is the agent in most of the refractory cases. In a fair

proportion, however, the disease is due to the large-spored fungus, and while such yield much more rapidly in most instances, still there are cases due to this variety of fungus which also prove persistently obstinate. Whichever fungus variety is the causative factor, the treatment remains the same. As yet we know that the recognition of the fungus variety has a bearing simply upon the prognosis as regards the probable time for a cure.

Strong remedies must, as a rule, always be used in the treatment of scalp ringworm in institutions. There are cases, it is true, in which the inflammatory element is, at first at least, prominent, and milder applications are usually demanded. In such cases, indeed, this inflammatory element may be said to be a natural attempt toward casting oft the disease. In almost all cases, sooner or later, however, the strong remedies are to be prescribed. What are they? In a great measure, one might say that the selection depends upon the habit or the prejudice of the prescriber. Still there is considerable unanimity among those having to do with these chronic cases as to the several remedies possessing greatest value. My own experience emphasizes the usefulness of sulphur, naphthol, iodine, chrysarobin, and croton oil. Sulphur and naphthol are most valuable and appropriate for those cases involving a greater part of the scalp. Chrysarobin and iodine for circumscribed areas, and croton oil for those patches which have persistenly failed to yield to other remedies.

There are certain adjuvant measures which should, however, be mentioned first, before taking up the manner of application of the above several remedies. First of all the hair of the entire scalp should be kept clipped short—very short, not allowed to be longer than one-fourth to a half inch. The disease is then more easily watched, and any new spots detected in their incipiency and promptly treated. In extensive and extremely obstinate cases it is even desirable to have the scalp shaved every six or seven days. The spread of the disease to other parts of the scalp and to other children should be prevented, as far as this is possible, by certain routine measures. These measures can, as a rule, be carried out even when the active remedies are being used on the patches; much less rigorously, however, with the croton oil treatment. With this object in view, the scalp is to be washed every second or third day with warm water and a medicated sapo-viridis:

℞.—Precipitated sulphur . . . . . ʒj.
Naphthol . . . . . gr. xx–xl.
Sapo-viridis . . . . . ℥j.—M.
Sig.—For shampooing.

The lather should be permitted to remain on for five to ten minutes, as it has in itself an inhibitory or destructive influence upon the fungus. If there is fear of taking cold, the parts may be enveloped with some covering. The lather is subsequently thoroughly rinsed off, the scalp rubbed dry, and then a general remedial application made. The following is useful for this purpose:

℞.—Precipitated sulphur . . . . . ʒj.
Naphthol . . . . . gr. xxx.
Petrolatum . . . . . ℥j.—M.

Or, instead of this salve, a lotion may be used for this general application, consisting of:

℞.—Carbolic acid . . . . . ʒij.
Resorcin . . . . . ʒj.
Saturated solution of boric acid . . . . . Oj.—M.

This general ointment or lotion should be applied daily. It is possible that the salve is more effectual in preventing the dissemination of the spores, although it is not so agreeable. As an additional measure in preventing the spread of the disease to others, paper or any material worn as a lining within the ordinary hat, which permits of frequent destruction or washing, should be constantly worn; paper is preferable, as it can be destroyed daily, if necessary.

A word as to depilation of the affected areas. This is a measure of some importance, if carefully and repeatedly done, but if the disease is at all extensive or the patient very young, it becomes almost impracticable. As an efficient substitute for this, and also possessing therapeutic properties of its own, I can cordially recommend a good depilatory powder. The following, if properly and freshly prepared of good materials, will answer the purpose:

℞.—Barium sulphide . . . . . ʒij.
Zinc oxide and powdered starch . . . . . āā ʒiij.—M.

At the time of application enough water is added to a sufficient portion of this mixed powder to make a paste, and it is then spread in a thick layer on the areas, slightly overlapping the edges; this is to remain on for several to fifteen minutes, according to the character of

the hair and the sensitiveness of the skin, or until heat of the skin or a burning sensation is felt; it is then washed off thoroughly, and if it has acted as it should, the hair of the patch, including the stumps will have been destroyed deep into the follicles, and possibly to the full depth of the latter. Should there be accidentally much resulting irritation a soothing ointment may be applied for a few hours or so; as a rule this is never necessary. The depilatory should be applied every five to ten days, depending upon the rapidity of regrowth. It should never be applied to an actively inflammatory patch.

As to the remedial applications to the individual areas, the same ointment as above, with double the quantity of sulphur and naphthol, will prove valuable in recent cases, and especially in very young subjects. Occasionally this amount of naphthol, in those of extremely sensitive skin, gives rise to a feeling of considerable burning, and in such cases this ingredient can be reduced in quantity. As a rule, however, the feeling of burning is over in a few minutes, and no active irritation follows. Exceptionally, also, the quantity of sulphur must be lessened in young children. This ointment can also be satisfactorily employed at the beginning treatment in extensive cases, the more recently affected parts usually yielding, leaving the chronic areas for the stronger remedies to be mentioned.

Ordinarily the best application for the patches in young patients and in recent areas in other cases is iodine tincture, containing a small quantity of mercuric iodide, as in the following:

℞.—Mercuric iodide	gr. j–iij.
Tincture of iodine	℥j.—M.
Make solution.	

I am convinced that the value of iodine tincture applications in this disease is much underrated. The preparation is painted on twice daily, two or three coatings at each time, till the areas become somewhat tender or till the film thus formed cracks or begins to loosen. The parts are then anointed with the milder of the above salves, and as soon as the film is detachable it is picked or pulled off. If there is active underlying irritation, which is not usually observed, a mild ointment may be applied for a day or so. The depilatory powder, if required, is then again used, and the paintings resumed. This is continued till the new-growing hairs show no evidence of fungus, and then the treatment is discontinued, and the patient kept carefully under

observation for several weeks. Should distinct signs of the disease again present themselves—if of doubtful character their significance should be decided by the microscope—the paintings are to be resumed. Or if the iodine applications seem to be slow in bringing about complete cure, another plan is to be instituted.

Chrysarobin is by far the most valuable application in most of the cases. It is made by various manufacturing chemists, and differs considerably in quality; naturally, an efficient preparation is an essential for success. It may be used in all cases, but more especially in cases of somewhat limited extent; it must be employed with greater care in patients under the age of three years. In most of the younger patients, in fact, the sulphur-naphthol salve or the iodine paintings will suffice to bring about a cure, and are to be preferred in such cases. Chrysarobin is most satisfactorily applied as a solution in chloroform :

℞.—Chrysarobin . . . . . q. s.
Chloroform . . . . . ℥j.—M.
Make saturated solution.

The areas are painted over with this till well coated with a film of chrysarobin, the chloroform rapidly evaporating. Over this is then painted three or four layers of good collodion. The plain collodion is a little too brittle, and the flexible collodion scarcely compressible enough; a mixture of equal parts is more satisfactory. In eight or ten hours the collodion is again applied. No further application is to be made till the film so formed begins to crack or break away, or begins to loosen, which it usually does in two to four or five days. It is usually loosened by the growing hairs beneath. As soon as it begins to become detatchable it is gently pulled off, and if there is any active irritation beneath, a mild ointment may be used for a few hours or a day till this is subdued. If there is any sign of stumps or growing hairs, the depilatory is again to be used, and the paintings with the chrysarobin and collodion resumed. This plan is to be continued, as with the plan with the iodine paintings, till the disease is cured, or till it is evident that stronger remedial applications are essential for a final result. Usually, however, the disease gradually yields to this drug, or, at all events, to the extent of leaving but an area or so for the croton oil treatment.

In some instances, but if used with care not in many, chrysarobin gives rise to a mild or moderately severe dermatitis of the surrounding

skin, and under these circumstances it becomes necessary to suspend its use temporarily. In rare instances this tendency to dermatitis may repeat itself, the skin of the patient being intolerant to this remedy, and it must be then set aside and give place to another plan. Such idiosyncrasy is, however, rare. If the patient or attendant is careless, some of the chrysarobin may be carried to the face or eye, and a temporary disturbance of these parts brought about. This rarely happens, however, with the plan of treatment as here advised, as the remedy is kept covered with the collodion film. If chrysarobin is used in ointment, as sometimes prescribed, this accident is much more likely to happen. In addition, the wearing of a paper cap or lining, as suggested, constitutes also another measure of safety against these effects.

In some instances these several methods will fail to bring about a cure; or more frequently will cure most of the areas, but fail to make sufficient impression upon a few patches. It is just in such instances especially that croton oil has its particular field of usefulness. In those cases, too, and in which, for various reasons, a rapid cure is desired, recourse may be had to this application. It is a severe remedy, and the parts are made actively inflammatory. Indeed, the object in view is to bring about an artificial kerion—a somewhat boggy and pustular inflammation, which, upon abatement usually results in the cure of the patch. In such cases as here mentioned, the application is most valuable, and, while it may in some instances destroy groups of a few follicles here and there in the areas thus treated, the subsequent growth of hair covers up these small points of baldness completely, so that this possibility is practically of no importance. The careless use of the remedy, and the pushing of the inflammatory action beyond a reasonable limit would, of course, result in considerable follicular destruction, and should be guarded against. If carelessly used, or its repeated application be unnecessarily long continued, complete baldness of the area could result, but this has never occurred in my experience; and it seems to me that such disaster could only be from gross carelessness, or possibly from some inherent peculiarity of the patient's skin. The scalp will fortunately stand a good deal of even reckless treatment, and yet finally show but little trace of it. Croton oil is, however, a strong remedy, and must always be used with caution. For this reason it is never to be employed in very young children. The application should be made by the physician himself,

or by a trained attendant. With this plan of treatment the use of the depilatory, exeept as a first preliminary, may be omitted.

At first it is desirable to apply the oil weakened with two or three parts of almond oil, and if it is found that no active inflammation arises, it should be applied stronger. In most cases, especially in older children, the pure oil is required. It should be scantily used, as the oil seems to have a distinct tendency to spread beyond the part to which it is applied. It is to be applied two or three times daily, and to bring about the desired amount of inflammation reaction usually two to six days are required. When it is possible to have poultices repeatedly applied afterward, the oil applications may be discontinued as soon as slight swelling and minute pustulation present; the subsequent poulticing will bring about sufficiently pronounced inflammation. In most cases, however, I have continued the oil applications till this latter condition was reached, and then applied boric acid ointment till the inflammation subsided. This ointment or the mild sulphur-naphthol ointment can then be used till the new hairs appear, and then all applications be discontinued; at the end of several days or a week, the parts and new-growing hairs are examined. If there are still signs of the disease the same method is to be repeated. One should be sure, however, that the disease still exists, before resuming the treatment. If the inflammatory action and pustulation have been quite of sufficiently high grade, a repetition is not usually necessary. If there are several patches to be treated, not more than one, if a large one, or two, if small, should be treated at one time. In fact, the oil should not be applied to an area exceeding, at the most, one to two inches in diameter; larger areas can, if desired, be treated in sections. As soon, however, as the inflammation has subsided a new area can be attacked.

In some cases, the disease is disseminated—disseminated ringworm of the scalp—consisting of scattered, small, pea-sized areas presenting five to ten diseased hairs or hair-stumps. Such cases can be treated by active methods—iodine or chrysarobin paintings. If the spots are numerous, the number may be usually brought down by the use of the sulphur-naphthol ointment, and then the remaining obstinate spots can be treated with the iodine or chrysarobin; if necessary for final cure, the oil can be employed. In such cases many of the areas yield readily with any good treatment, leaving behind several or more obstinate spots.

In those instances of scalp ringworm in which the disease is so extensive as to practically involve the greater part of the scalp, presenting large and irregular areas or confluent sheets, it is advisable to use the stronger sulphur-naphthol ointment till the disease is reduced in extent. Or this plan can be used, and small portions treated with the iodine or chrysarobin paintings. Croton oil, if used at all, should be left till the disease is reduced to several circumscribed areas; it is never to be used in the beginning of treatment in these confluent cases.

Constitutional treatment is not usually deemed of any value in ringworm cases, the cause being purely local; but in these hospital patients I have always believed that the administration of cod-liver oil and iron, especially the former, have an influence for good, directly upon the general health, and indirectly upon the cutaneous disease—in rendering the "soil" a less desirable one for fungus vegetation, and in this manner lending some aid, although doubtless very slight, toward the final cure. It has seemed to me, too, that sulphur, internally administered in small doses, by its exhalation through the skin, makes the structure a less desirable habitat for the fungus, and thus be of contributory value.

EXTRACTION OF COMPLICATED CATARACT; COLLAPSE OF BALL; INJECTION OF SALINE SOLUTION; RECOVERY.

By HOWARD F. HANSELL, M.D.

The recommendation of Dr. Herman Knapp, who reports three cases,[1] and of Dr. J. A. Andrews, who, at Dr. Knapp's suggestion, published his experience,[2] encouraged me in the following case to resort to the expedient of injecting sterile salt solution, when, after the extraction of a milky-white fluid cataract, the ball collapsed and the operation almost surely presaged failure.

A man, aged fifty-six years, was admitted into the ophthalmic ward of the Philadelphia Hospital on account of blindness in the left eye, of one year's duration, and imperfect vision in the right eye, from corneal opacities the result of old severe ulcerations. He was a large, apparently able-bodied man whose health had been undermined by idleness, syphilis and intemperance, and like so many in the same social position, he was devoid of courage and will-power. The right eye was free from active inflammation. The iris reacted well, the lens was clear, and there was no disease of the fundus. In the left eye the cornea had lost some of its transparency from previous keratitis, the anterior chamber was abnormally deep, there were no attachments between the iris and the lens capsule, the lens was dense, white, milky, homogeneous and showed no striæ between the sectors; light projection good.

Extraction of left lens was proposed and done. The usual antiseptic precautions preceding and during operation were observed. While making the corneal incision, the patient, by forcible compression of the lids, dislodged the speculum, and before the cut was complete expelled a small portion of fluid vitreous together with the aqueous. The speculum was allowed to remain because its removal would have necessitated withdrawal of the hand that held the knife, and the upper lid was retracted and held by an elevator. After the incision, which was made in the corneal limbus, was completed, the iris was prolapsed by the escaping vitreous. By this time a large portion of the latter had been lost. The prolapsed iris was cut off and the lens removed with the Levis wire loop. After the lens was free from the ball it was found to be adherent to the vitreous, and could be detached only after some effort. The sclera was collapsed and the corneal lip everted. Warm sterile salt solution was injected with a curved small-nozzled eye-dropper

[1] Archives of Ophthalmology, No. 3, 1899.
[2] Archives of Ophthalmology, No. 1, 1900.

until the ball had resumed its rotundity. On account of the leaking of the mixed vitreous and salt solution the edges of the wound could not be brought into apposition by repeated injections, and could only be restored to their proper place by manipulation. A binocular bandage was applied and allowed to remain twenty-four hours. Fortunately, the patient was operated upon in his own bed, so that he was not subjected to further loss of vitreous by the physical exertion of moving. At the expiration of twenty-four hours the bandage was removed, the lower lid retracted, boric acid and atropia instilled, and the eye closed for forty-eight hours. Recovery was uninterrupted, but slow. Four weeks after operation signs of irritation had dissappeared, to recur only after examination. Vision equalled 10/200. Were it not for the corneal opacities it would probably be four times as acute. With a strong spherical glass the patient can readily tell the time on a small watch. The pupil is black, the vitreous clear and the fundus healthy.

I have ventured to report this operation, and its result, and to call attention anew to the great value of the injections of sterile salt solutions (probably any aseptic unirritating fluid would serve as well) in saving useful vision in eyes, that without them would almost certainly perish. I am aware that the loss of very considerable vitreous is not incompatible with good recovery, but am well convinced that in desperate cases the injections contribute in no small degree to a favorable result, and are extremely valuable.

My colleague, Dr. de Schweinitz, described before the Section on Ophthalmology, College of Physicians, April, 1900, his happy experience in saving an eye, and in giving good vision by substituting salt solution for the lost vitreous. Notwithstanding the inversion of the entire iris and the manipulation necessary to extract the lens and restore the parts to their proper relations, the eye recovered with 20/50 of normal vision.

Dr. Knapp reported two cases:

Mr. W., aged fifty-two years, had had a cataract extracted from the right eye, three years before, with good result. In the left the iris was tremulous, and the opaque lens oscillated backward and forward, and the ball was slightly softer than normal. After making the usual corneal section, fluid vitreous escaped freely and the lens was expelled with difficulty, and so much vitreous was lost that "the scleral capsule lay folded together like a piece of wet linen over which, in the upper part, the cornea projected like the visor of a cap." Four small syringefuls of a warm sterilized 7 : 1000 saline solution were injected into the globe, filling it so that the lips of the wound were brought into apposition. In forty days: filled and tension normal, eye-ground easily seen and healthy, vision 20/100. In Case II, the vitreous escaped after expulsion of the lens, and one syringeful of saline solution only was required. Healing was complete on the tenth day. Vision equalled counting fingers. "Thus far, only an operative success."

Dr. Andrews' case was remarkable not only in restoring the shape to the ball, and the proper relation of the ocular contents, but in giving final vision of 20/30. "I should say that more than half of the vitreous escaped through the wound. The whole eyeball collapsed. I injected into the globe a quantity of warm sterile solution of sodium chloride (6 : 1000). The recovery was smooth and uneventful."

In view of the testimony of these two published cases and the results in de Schweinitz's case and in my own, I feel justified in advocating the further use of the saline solution in cases of collapsed eyeballs following loss of vitreous in cataract extraction and in traumatism. The instrument through which the injection is made seems to me to be immaterial. The ordinary eye-dropper with a fine curved nozzle answers all requirements of the more expensive Myer's syringe, but in its use care must be taken to avoid suction of the ocular fluids by preventing expansion of the rubber bulb while within the limits of the ball.

A FEW INTERESTING CASES OCCURRING DURING MY LAST SURGICAL SERVICE.

A Case of Appendicitis; Profuse Hemorrhage following the Tearing of an Adhesion whilst Removing the Appendix.—Tubercular Peritonitis Simulating Abscess of the Liver.—Strangulated Umbilical Hernia, with Localized Peritonitis and Paralysis of the Bowels; Relieved by an Operation, together with an Injection of a Saturated Solution of the Sulphate of Magnesia Introduced Directly into the Small Intestine at the Time of the Operation.—Elephantiasis of the Penis.

By ORVILLE HORWITZ, M.D.

A CASE OF APPENDICITIS; PROFUSE HEMORRHAGE FOLLOWING THE TEARING OF AN ADHESION WHILST REMOVING THE APPENDIX.

I was sent for late in the evening of April 22d to examine a patient who had just been admitted to the surgical ward, who gave the following history:

He was thirty-eight years of age, a bricklayer by occupation. Two days previous to his admission to the institution, whilst at work, he was suddenly seized with a sharp pain in the right side, which later on was attended with nausea and vomiting. The bowels were constipated. The pain had continued ever since his admission, with a tendency to increased severity. On examination the abdomen was found to be slightly distended and rigid, especially on the right side. There was marked tenderness on pressure over the right iliac fossa, and a slight indurated mass could be distinctly felt in that vicinity. The pulse was 118; temperature, 102.

He was placed on the internal use of salines and an ice-bag was applied locally. At the morning visit I found that, in spite of the fact that there had been two movements from the bowels, the patient was not so well, the local symptoms having increased in severity. I decided on immediate operation. On opening the abdominal cavity, over the appendix, a circumscribed abscess was found containing about one ounce of pus; after this had been evacuated the appendix was seen to be bound down to the head of the colon, the tip being adherent to some point near the bottom of the pelvis. The meso-appendix containing the appendicular artery was ligated and cut, and the appendix gradually separated from its attachment, with the exception of a slight adhesion at the extremity. An effort was made, without success, to discover if it were adherent to any important structure.

The index finger of the right hand was inserted back of the adhesion and it was slowly and steadily dissected loose. No sooner was the end of the appendix free from the attachment than a jet of dark-blue blood about as thick as the little finger began rapidly to flow, instantly filling the cavity. Iodoform gauze was packed tightly into the wound and the bleeding temporarily controlled by pressure. The bloodclots were now washed away by means of a warm salt solution, and an effort made to find, if possible, what vessel had been injured. As soon as the gauze packing was disturbed, even in a slight degree, the cavity again became filled with blood, so that it was impossible to determine exactly what vessel had been wounded. From the quantity of blood which issued from the orifice of the injured vessel it was presumed to be a large branch of the internal iliac vein. The gauze packing was replaced in the wound and the cavity again freed from blood, but it was found that there was a great deal too much oozing going on around the gauze to leave the vessel, with safety, unsecured.

Accordingly, the edges of the wound were held as widely asunder as possible by means of blunt retractors, and the assistant was instructed to slowly and gradually remove the gauze packing, starting with the edge nearest to the operator. As the packing was removed I followed it closely with a pair of hemostatic forceps; after a small portion of the gauze had thus been removed, I observed a spot under one edge of the packing from which the blood was beginning to flow freely. I directed the assistant to desist from removing the packing and carried the forceps well down under the gauze and below the bleeding point; the hemorrhage was controlled, and on removing the gauze I discovered that I was fortunate enough to have secured the vessel. On examining the depth of the wound I discovered another point bleeding, evidently the site from which the adhesion had been torn. This was also secured by hemostatic forceps. An effort was then made to cast the ligature below the hemostatic forceps, but this was found to be impracticable, owing to the want of sufficient tissue wherewith to hold the ligature. It was, therefore, decided to leave the forceps *in situ*. The cavity was packed lightly with iodoform gauze and the forceps left protruding from the wound. An effort was made to remove the gauze on the third day, but it was so adherent and caused so much pain that it was deemed advisable to leave it until it would naturally become loose. This did not take place until the sixth day, at which time both the packing and the forceps were removed, the patient making an uninterrupted recovery.

Should I have found that packing had not controlled the hemorrhage, or had I not been fortunate enough to have secured the bleeding vessel by means of the forceps, I should have directed the assistant to have made pressure with his finger on the bleeding point, and have ligated the common iliac vein.

Cases have been reported in which the appendix has been found adherent to well nigh all the pelvic and abdominal vessels, as well as the iliac bloodvessels. Whenever this condition exists severe hemorrhage is very apt to ensue on breaking up the adhesions; the operator, being aware of this fact, should always be on his guard when dealing with an appendix adherent to any of the surrounding structures.

The complication just recounted is a rare one; but any surgeon is liable to encounter it at any time, and may not be so fortunate as to secure the vessel as I was.

For a time it looked very doubtful, both to the onlookers and to myself, whether the patient might not succumb to the loss of blood, the hemorrhage was so profuse. It was impossible to say what vessel was injured; I do not know now. It was difficult to locate exactly the bleeding spot, and I am convinced that had I not been successful in grasping the vessel in the bite of the forceps I could not have controlled it by pressure. It is interesting to note that both the gauze and the forceps were left in the bottom of the cavity for six days without producing any untoward symptoms.

TUBERCULAR PERITONITIS SIMULATING ABSCESS OF THE LIVER.

In May last I was requested by Dr. Sallinger to see, in consultation, a patient from the medical ward, who presented the following clinical history:

He was a negro, thirty-three years of age, a laborer by occupation, who stated that he had been living in the South previous to his coming to Philadelphia. When I first saw him he had been in the medical ward of the hospital for two months. At the time of his admission he stated that he had had chills, and that he was subject to attacks of chills and fever. His temperature was 103°. He complained of aching in the back and limbs; there was some vomiting, and the bowels were constipated. On admission his case was regarded as one of probable malaria; an examination of the blood failed to reveal anything that would verify this conclusion. Under treatment the patient seemed to gradually grow worse; the temperature assumed a typhoid type, being often at night as high as 103°, and down to 100° in the morning; the tongue was dry and coated; there was bilious vomiting at times, associated with a good deal of diarrhœa. The movements not being typhoid in character, the case was regarded as one probably that would develop into enteric fever. This condition remained unchanged for a period of about four weeks longer. There was still continued fever, occasionally rigors and free sweats, at times vomiting and diarrhœa; gradually jaundice supervened, as shown by the conjunctiva and the urine; there was pain in the right shoulder and back. The urine contained bile pigment, trace of albumin, low percentage of urea, no casts. The liver was slightly increased in size and seemed sensitive to pressure. Heart and lungs normal.

Twenty-four hours after the first visit I again saw the patient, in consultation with Drs. Sallinger and Barton, and found him in about the same condition, but in addition he was slightly delirious, the delirium being of a muttering type; he was, however, capable of being aroused. The abdomen was somewhat distended and tympanitic.

Firm pressure over the liver seemed to produce pain; the organ being somewhat enlarged, and imparted to the finger the sense of pseudo-fluctuation.

It was obvious that some abdominal mischief was going on, and we were all inclined to believe, from the clinical history of the case, there was probably an abscess of the liver.

An exploratory laparotomy was decided upon. An incision was made on the right side over the liver and the abdominal cavity opened, when it was found that the individual was suffering from a tubercular peritonitis, with a somewhat congested and enlarged liver; no sign of abscess could be detected. The abdominal cavity was flushed with a hot salt solution, dusted with sterilized iodoform, and an iodoform-gauze drain inserted. The patient died on the fourth day, apparently from exhaustion. No autopsy was permitted.

In analyzing the history of this interesting case, it will be observed that during the last three weeks of his life all the symptoms pointed to abscess of the liver; a positive diagnosis without an exploratory operation, which did neither good nor harm, was out of the question.

Incision and drainage in cases of tubercular peritonitis are now recognized as the proper methods of treatment, and it is a question if this procedure had been entertained earlier whether benefit would not have followed in the case here recounted.

In Volume III of the PHILADELPHIA HOSPITAL REPORTS, I recounted at length a case, seen in consultation with Dr. Packard, of tubercular peritonitis which simulated appendicitis. An exploratory abdominal incision was made, the true condition discovered, the abdominal cavity irrigated and drained, the patient making an excellent recovery.

A CASE OF STRANGULATED UMBILICAL HERNIA, WITH LOCALIZED PERITONITIS AND PARALYSIS OF THE BOWELS; RELIEVED BY OPERATION, TOGETHER WITH THE INJECTION OF A SATURATED SOLUTION OF THE SULPHATE OF MAGNESIA INTRODUCED INTO THE SMALL INTESTINES AT THE TIME OF OPERATION.

The patient was an Irish woman, about forty years of age. On admission to the hospital she stated that she had been afflicted with umbilical hernia for the past six years, which had gradually increased in size, and was occasionally the seat of pain. For the last two years she had been unable to return the contents of the hernia into the abdominal cavity, For three days before coming to the hospital she had suffered from obstinate constipation; large doses of purgative medicine which had been taken had failed to bring relief. The hernia was tense, painful, and tender to the touch, followed by nausea and occasional vomiting.

On admission the temperature was 102°, pulse 122, respiration 33. Tongue dry and coated; vomited bile occasionally; the bowels had not been moved for three days. The abdomen was enormously distended, tender to touch; abdominal muscles tense. A hernia protrusion about the size of a fœtal head, composed of intestine and omentum, occupied the position of the umbilicus. It was incarcerated, tense, and tender on pressure. The skin over the surface of the tumor

was of a deep dusky-red hue, and looked as if it were about to become gangrenous.

An anæsthetic was administered, and an effort made, by taxis, to reduce the hernia, without success. An immediate operation was decided upon.

An elliptical incision was made at the base of the tumor, and all the congested skin and connective tissue covering the hernia removed, exposing the sac, which was next opened in the median line. It was found to be thickened and inflamed. The sac contained enormously distended and inflamed intestine, together with omentum. The bowels and omentum were adherent to each other, as well as to the sac, the adhesions being separated with great difficulty. A marked constriction was found at the neck of the sac, which was relieved in the usual manner. After dividing the constriction it was found that there was a good deal of localized peritonitis present, the bowels being so much distended with gas that it was almost impossible to return them to the abdominal cavity. In order to relieve this condition and to overcome the paralysis of the intestines, the expedient suggested by Dr. Andrew J. McCosh, of New York, was adopted. A small hollow needle was inserted into the small intestine, and the accumulated gas allowed to escape; then, by means of a syringe, two ounces of a warm saturated solution of the sulphate of magnesia were injected into the bowel, the needle withdrawn, and the wound closed by means of a Lambert suture. The abdominal cavity was flushed with a hot normal saline solution and the abdominal wound closed in the usual manner.

Under the circumstances it was considered safest to employ drainage; accordingly an incision was made in the right iliac fossa, and a glass tube introduced, which was removed on the third day.

The large quantity of serum discharged through the drainage tube for two days succeeding the operation showed that its employment was a proper expedient. It is a rule with me to employ drainage for a couple of days after an abdominal operation where there has been extensive adhesion.

As soon as reaction had fairly set in and the stomach was quiescent, a ten-grain dose of calomel was administered. Rectal alimentation was relied upon for the first thirty-six hours; after which time the patient was fed by the mouth. Five hours after the operation the bowels began to move freely, and immediately the abdominal distention, pain and tension began rapidly to subside. With the restoration of the normal function of the bowels the nausea and vomiting ceased.

The point of interest in this case is the effect of intestinal injection of a saturated solution of the sulphate of magnesia for the relief of the paralyzed condition of the bowel. I have employed the method in three somewhat similar cases and always with marked benefit. As McCosh has pointed out, in the *Annals of Surgery* for June, 1897, this method of employing the sulphate of magnesia is especially serviceable in cases of septic peritonitis where great distension exists.

A large dose of calomel in paralysis of the bowel, with marked distention, is always beneficial; not only does it have a tendency to induce active peristaltic action, but it is an excellent diuretic, having a tendency to prevent suppression of urine which sometimes accom-

panies abdominal complications. Small doses of the mild chloride of mercury, repeated at short intervals, will not produce the active vermiculation which is obtained by the administration of a single large dose of the remedy.

ELEPHANTIASIS OF THE PENIS.

Elephantiasis is rarely met with in America or Europe, but sporadic cases are occasionally seen.

In the tropics, however, it is often found, and is frequently endemic to certain districts, attacking the organs of generation of both sexes, next in frequency after the lower extremities.

The disease appears to be a hypertrophy of the fibrous tissue of the skin and subcutaneous connective tissue, attacking the last-named structure first. This is followed in time by an increase in the size of the neighboring organs, disturbing the circulation, and giving rise to chronic inflammation of the lymphatic vessels of the part.

It is very unusual for elephantiasis to attack the penis primarily, but it frequently follows involvement of the scrotum.

Dr. Then, in the *Transactions of the London Pathological Society*, 1880, gives a case of elephantiasis of the penis where no appearance of a multiplication of cells by division could be detected under the microscope, and hence infers that the whole of the cells are derived from the white blood-corpuscles.

Lewis, Bancroft, Mansem and Henry have of late years pointed out that elephantiasis, if not caused by, is at least frequently associated with, the presence of a parasite, the falaria sanguinas hominis, of which there are three varieties: Falaria diurna, falaria nocturna and falaria perstans. Of these three divisions it is probable that the falaria nocturna which gives rise to certain forms of elephantiasis, as well as the conditions known as lymph-scrotum and hæmatochyluria, are the most common. The embryo in tropical cases is present in the blood in large numbers at night, and almost entirely absent during the day. Stephen Makenzie asserts that if the patient sleeps during the day and is awake at night, the condition is reversed. Osler states that these parasites cannot be found in every case of elephantiasis, and reports two cases coming under his own observation where falaria in the exuded fluid or in the blood at night could not be detected. He further observes that the majority of cases of elephantiasis which occur

in this country are non-parasitic, whilst the directly opposite condition pertains in cases occurring in China. The parasite is found principally in tropical climates, and according to the observations of many American writers, it exists extensively in the Southern States. The falaria sanguinis hominis appears in the blood in its embryonal form, and is fully developed only in the lymphatics.

The scrotum is more frequently the seat of the disease than the penis, this organ as a rule being affected secondarily. Two cases are reported involving the penis alone, one by R. W. Taylor and the other by R. F. Weir, of New York. In Taylor's case the patient was a young Hebrew in whom the condition followed an injury to the organ. In Weir's case the hypertrophy followed a stricture of the urethra associated with an abscess resulting in a urinary fistula.

The history of the case which came under my care is briefly as follows:

The individual was a colored man, forty-five years of age, sailor by occupation. Family history, negative. Patient states that he never had any venereal disease. About six months before coming to the Philadelphia Hospital, while at sea, having abstained from sexual intercourse for four months, he noticed a small, slightly elevated, hard lump, about the size of a pea, on the left side of the frenum. This lump increased slightly in size, became irritable and ulcerated at the base, from the necessary friction produced by coming in contact with the clothing. Gradually sloughing set in, until the tumor hung by a strip of skin, which he cut through with a pair of scissors; the resulting raw surface healed rapidly. About three weeks later the entire penis began to enlarge until it gradually reached its present dimensions. He has never had any pain or experienced any difficulty in urination. He has lost slightly in weight.

On examination, the glans penis was small and almost entirely hidden by a firm fibrous mass which entirely surrounded the end of the organ. Between the penis and scrotum there is a distinct line of constriction, the skin of the latter being perfectly normal. The left testicle was easily discovered, but the right testicle could not be found, and was supposed to have undergone atrophy. The skin of the penis was cut up into furrows running longitudinally, which were crossed by others running more or less obliquely, dividing the organ into lobules, which were hard, firm and elastic. From pubis to the glans penis, along the dorsum of the organ, the measurement was eleven inches. The circumference of the mass in its thickest portion was nine and one-half inches. On palpation a distinct doughy sensation was imparted to the touch, but there was no pitting on pressure. A very careful study of the blood was made by my colleague on the staff, Dr. F. P. Henry, but the falaria sanguinis hominis could not be detected.

The patient's temperature was normal. Sexual power was completely lost. Examination of the urine, negative.

The patient was etherized and an incision was made along the entire length of the dorsum of the penis, being about ten inches in length. The skin was found to

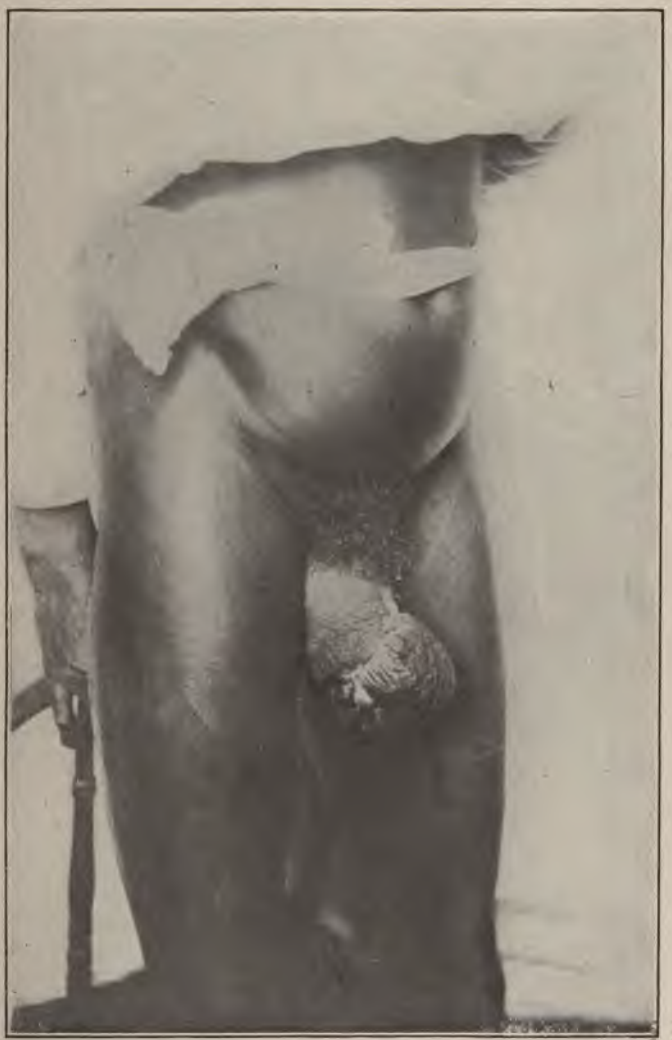
be tough and fibrous, and on division a thick, white, elastic fibrous tissue was exposed; it was impossible at first to identify either the corpus cavernosum or spongiosum. To guard against wounding the urethra a small-sized bougie was passed into the bladder. The tough fibrous tissue was then dissected entirely away from the penis, when a strong, thick band was bound, passing along the entire length of the under surface of the organ, which was firmly attached to the central tendon of the perineum. On removing the fibrous mass from the vicinity of the base of the penis, the missing testicle was found, pulled up out of place; it was dissected loose and replaced in the scrotum. The hemorrhage, which was not so profuse as had been expected, was easily controlled by means of hemostatic forceps and very few ligatures were required. After the operation an ordinary antiseptic dressing was applied and the body of the penis allowed to granulate. The complete healing of the wound occupied four months, at the end of which time the patient had entirely recovered. On examination after recovery it was found that the newly-formed skin was tightly adherent to the body of the organ, which held the penis in a horizontal position. The patient stated that he was in perfect health, and that his sexual powers were completely restored.

A brief extract from the interesting pathological report made by Prof. H. F. Harris, is herewith appended:

"On microscopic examination the epidermis covering the diseased area is found greatly thinned, and the epithelial ridges are almost entirely destroyed. Here and there, however, thin rods of epithelium, which are evidently the remains of these ridges, project down for a short distance into the true skin; the cells of which these rods are composed contain much almost black pigment, and they do not react to either basic or acid aniline dyes. Just beneath the epidermis there are numerous rounded masses of more or less entirely keratinized epithelial cells. They are sometimes in contact with the epidermis, but more generally seem to be quite free in the derma proper, without any connection with the epithelial layer. While the cells of which they are composed are in most instances keratinized, in some cases the cells which form the outer boundaries of the masses still preserve the morphologic and staining peculiarities of the younger cells; sometimes those cells are piled upon each other several deep. The epithelial cells of the epidermis, as a rule, preserve their normal size, shape and general relation to each other, but they do not stain as readily as normal cells. In addition to this, many cells in the prickle layer are swollen to twice the normal size. The protoplasm of these cells is homogeneous and takes acid stains faintly, the nuclei stain feebly or not at all. Occasionally, a leucocyte can be seen between the cells. The layers of cells which form the deeper portions of the malpighian layer are almost black from the presence of a dark-brown pigment; the pigment is so dense that the peculiarities of the cells in this situation cannot be made out with certainty.

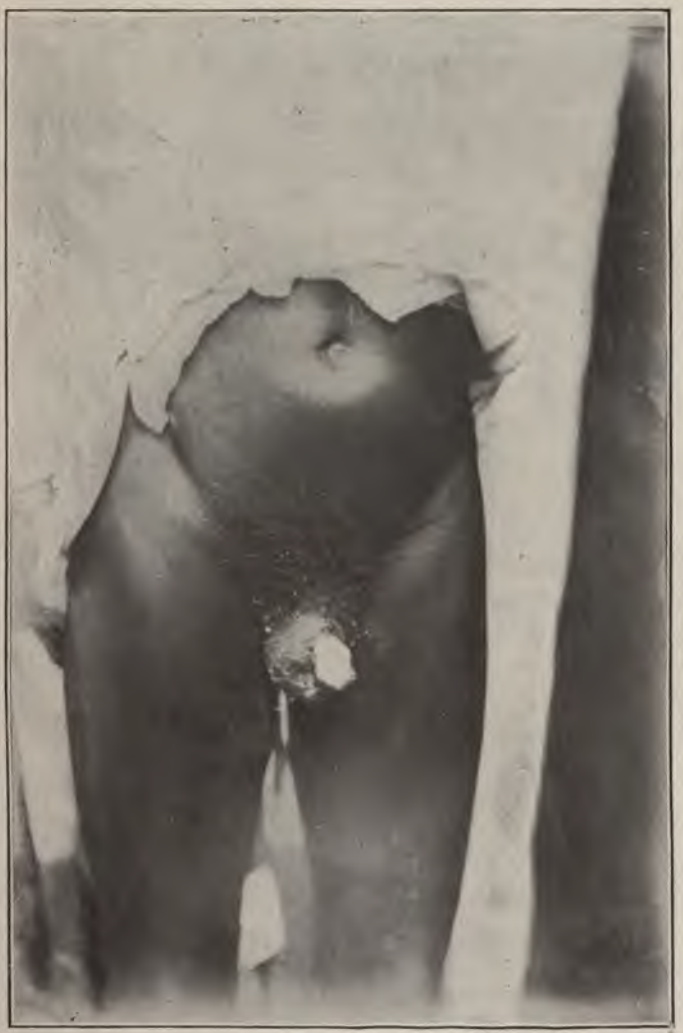
"The greater part of the tissue is evidently from that part of the penis upon which no hair occurs, but in sections from one of the pieces a few were observed. No changes in the shaft could be made out. The cells of the inner cells of the outer root-sheath are plainly in a state of degeneration; their nuclei stain faintly or not at all, and their protoplasm is faintly colored by the acid dyes. Even the outer cells of the outer root-sheath are elongated and their nuclei are very irregular in form. The cells of the sebaceous glands present more nearly a normal appear-

FIG. 1.



Elephantiasis of the penis before operation.

FIG. 2.



Showing result after removal of hypertrophied tissue in a case of elephantiasis of the penis.

ance than any of the other epithelial structures, but they are in many cases elongated and take stains poorly.

"Sweat-glands are only occasionally found. The coils are often separated from each other by dense masses of cells. These cells will be referred to later.

"The true skin is enormously hypertrophied. This is principally due to an increase in the amount of collagenous tissue, but not in an inconsiderable degree to the presence of collections of cells around the blood-vessels of this tissue. The collagenous tissue occurs in the thick bundles which are almost invariably disposed in planes parallel to the skin surface. In the deeper portions of the skin well-defined fibrils of elastic tissue are often found; they are, in general, run from the deeper layers of the skin toward the surface. At intervals through the tissue comparatively large, robust bundles of involuntary muscle fibres occur. They are not, probably, of a new formation, but result from the hypertrophy of the pre-existing muscle of the parts. In the true skin extending downward for a considerable distance there are numerous small, very dark pigment masses, generally of a rounded or irregular form. These granules may be seen in the process of formation from the lower layer of the epidermis.

"The blood-vessels are comparatively scant, but those which are present present interesting changes. Contrary to the observations of others, I have found the changes in the arteries much more pronounced than those in the veins. The alterations in the latter consist principally in a marked dilatation of their calibre; in addition to the endothelial cells lining, their inner coat shows a marked decrease in their power of taking stains, and, in some cases, they do not stain at all. Rarely the outer coats of these vessels show marked thickening, and, in almost every instance, are more markedly cellular than normal. The arteries are all small, and very frequently their lumen in encroached upon by thickening of their walls, and in these instances the intimæ are represented by a structureless hyaline membrane which takes the acid stain. The muscular coats are rarely so stained that their true nature can be recognized. Replacing the muscular coat in many of the vessels are collections of cells which have the appearance of lymphoid cells; sometimes these masses of cells exactly occupy the muscular area, but in other cases they encroach upon the intima and push it inward. The adventitive of the vessels are in most instances decidedly thickened and contain lymphoid and plasma cells. The entire walls of some of the vessels are hyaline; here the intimæ are generally swollen. Occupying the lumina of some of the arteries are yellow, entirely homogeneous masses which take acid stains, but no basic ones, and would appear to be hyaline thrombi. None of these were seen in vessels whose walls were hyaline, but were frequently observed in those the muscular walls of which were infiltrated with cells.

"The perivascular lymphatics were all greatly dilated.

"The perivascular lymphatics are almost always greatly dilated and contain collections of lymphoid and plasma cells; within these collections of cells, usually near their edges, mast cells frequently occur, but no polynuclear leucocytes were found.

"Between the bundles of collagenous tissue, and having no apparent connection with the lymphatics of the blood-vessels, are often collections of cells which in every way resemble those just spoken of; whether they are only a part of these masses cut so as not to sow the vessels or entirely separate them could not be determined. Scattered through the tissues generally plasma, lymphoid, and mast cells

are of frequent occurrence. The tissues are abundantly supplied with characteristic branched connective-tissue cells. None of these cells mentioned are elongated or twisted, as would be supposed to be the case had they been subjected to considerable pressure. A careful study of the section failed to reveal the presence of micro-organisms.

"Attempts to study the nerves of the tissue by Golges' silver method were unsuccessful; as is often the case, impregnation did not occur."

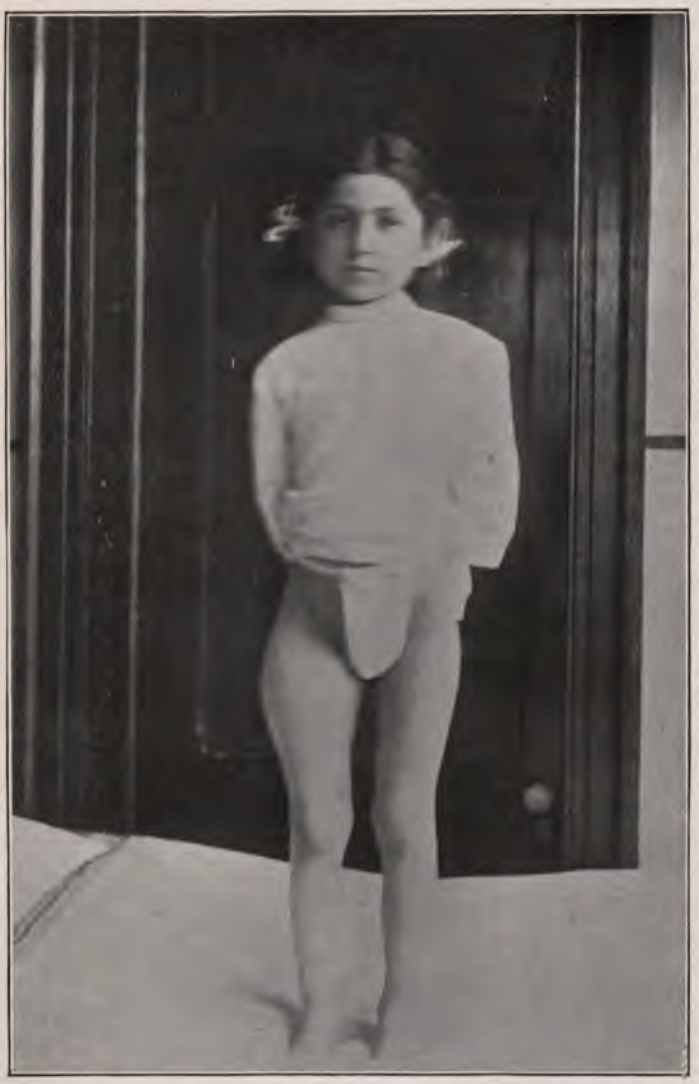
AMPUTATION AT THE HIP-JOINT FOR DIFFUSE TUBERCULAR OSTEOMYELITIS OF THE FEMUR.

By ALFRED C. WOOD, M.D.

The patient, a girl, six years of age, was taken to a hospital in May, 1896, on account of a swelling of the upper extremity of the right thigh. There were no facts in the family history that seemed to have any bearing on the case. The child had always had good health, she had not had typhoid fever, or any of the infectious diseases. There was no history of any accident. About one year previous to the above date, a swelling was noted on the antero-lateral aspect of the upper part of the right thigh. It was painless, free from any signs of inflammation, and apparently did not inconvenience the child in any way. The swelling had slowly but steadily increased in size up to the time of this note. There had been some pain recently which was worse at certain times than at others. A few weeks before coming under observation, the patient began to limp slightly in walking.

The general size and shape of the swelling are well shown in Figs. 1 and 2. The skin over the enlargement was natural in appearance, free from redness, and moved freely over the deeper structures. The superficial veins were not especially dilated, although slightly more prominent than on the opposite side. The mass was moderately tender on pressure. To the touch it was everywhere uniformly resistant. There was no superficial œdema and no fluctuation. The two conditions that seemed most likely to explain this case were osteo-sarcoma and deep abscess resulting from bone inflammation. The probabilities seemed more in favor of the former. The parents were advised to bring the child into the hospital for operation, as no form of palliative treatment promised any relief. Accordingly, the child was admitted on May 26, 1896. The general health seemed to be good, the organs were sound, the temperature was 100°, and the pulse, 120. The urine was normal. The operation was performed May 27th. Permission having been obtained from the parents, preparations were made for hip-joint amputation in case the exploration of the swelling revealed a malignant growth. Incision over the most prominent part of the enlargement showed normal superficial structures and the absence of a neoplasm, but disclosed a small collection of pus beneath the deep mass of muscles. The periosteum was raised from the femur, and the bone was carious. Surrounding the small fluid collection was a very thick, dense wall of lymph and œdematous infiltration. The diagnosis of subperiosteal abscess seemed justified, and counter-openings were made on either side posteriorly, in order to secure perfect drainage. After curetting the bone and the lining membrane of the cavity, the wounds were packed with iodoform gauze, and dressed in the usual manner. The child reacted well from the operation, and the subsequent dressing consisted in changing the gauze and irrigating the wounds with antiseptic solutions

FIG. 1.



Reproduction of photograph taken before first operation.

daily. The patient left the hospital July 17th, the swelling not having been influenced much, if at all, by the operation.

She was re-admitted July 30th, when it was learned that she had been to another hospital for treatment. The tracts had been allowed to close in the meantime. The circumference of the thigh at the affected part was sixteen inches, which was an increase of one inch since leaving the hospital. The skin over the swelling had now a slight livid hue, and the veins were more prominent. It was supposed that the closing of the external incisions, before the deep portion of the wound had healed, accounted for the aggravation of the symptoms. Accordingly, the former tracts were opened, July 31st, and a small quantity of pus evacuated. The cavity was thoroughly curetted and treated as before. The patient again left the hospital, August 12th. The wounds were being drained by means of gauze tampons, and dressed antiseptically daily, but without effecting any notable improvement.

In September, 1896, she came under the care of the writer at the Philadelphia Hospital. The swelling had increased rapidly in size, the condition at this time being well illustrated in Fig. 3. The use of the probe through the anterior sinus, shown in the cut, as well as in the two posterior tracts, revealed very extensive disease of the femur. The child now had considerable pain, and was unable to use the limb. Having full assurance that the best possible drainage had been given the diseased area for a considerable period, and, from my examination of the patient, it seemed certain that nothing short of removal of the femur would be of any avail, the parents accepted this advice, and the operation was performed September 26, 1896.

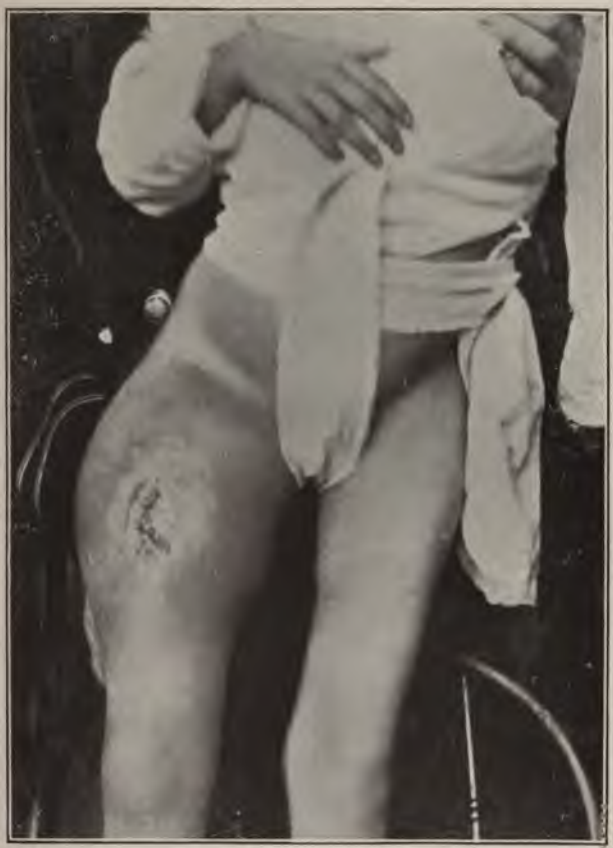
While the ether was being administered the limb was held perpendicularly in order to empty the vessels as thoroughly as possible of blood. It has been shown that after a certain degree of anæmia has been obtained by gravity in this manner, that there is a reflex contraction of the blood-vessels which empties them still further. The same result is thus obtained as by using the elastic bandage, which was inappropriate in this case, as it might have resulted in forcing septic matter into the system. The circulation was then controlled after the manner recommended by Dr. John A. Wyeth, of New York. As will be seen by a reference to Fig. 3, the diseased area extended so close to the trunk that a very high amputation was necessary. The flaps were composed of skin only. During the ex-articulation, although the limb was carefully held, the neck of the femur broke, leaving the head of the bone still in the socket. I was able, however, to complete the removal by grasping the fragment with the fingers and dividing the remaining portions of capsule and the ligamentum teres.

Nothing could have been more satisfactrry than the control of the hemorrhage. There was absolutely no oozing until the elastic band was removed, and at the some time, the pins kept the tourniquet so well in position that no difficulty whatever was experienced in removing the head of the bone after the neck had broken, a procedure which would have been nearly, if not quite, impossible without their use. The main vessels were caught and ligated, and the tourniquet removed, when the additional bleeding points were tied. As is always the case when operating through tissues the seat of chronic inflammation, these were very numerous. Although the operation was done with at least the usual deliberation, and I did not know that it was being timed, the following memorandum handed to me afterward by Dr. Myers, one of the resident physicians, is, perhaps, worth recording. The figures indicate the time that elapsed from the begining of the operation:

FIG. I.

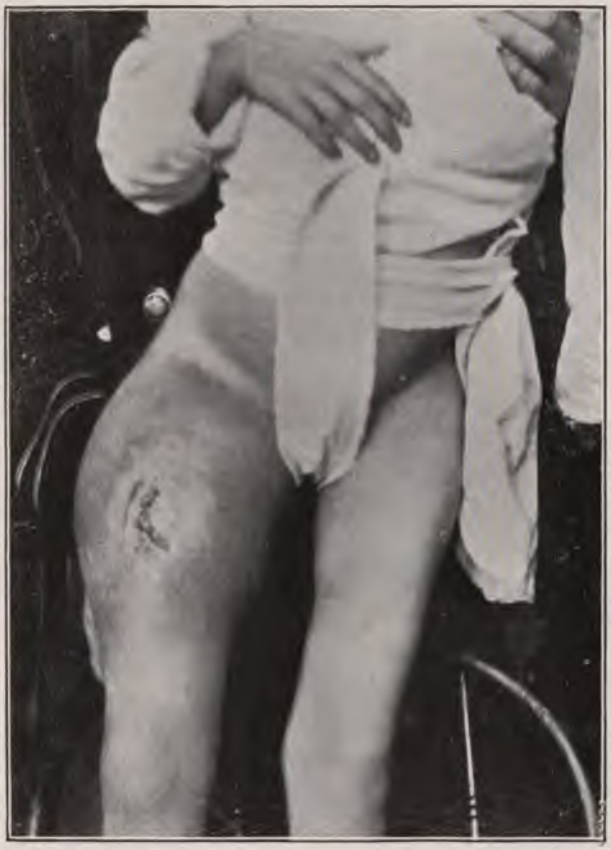
Reproduction of photograph taken before first operation.

FIG. 3.



Reproduction of photograph taken just before hip-joint amputation.

FIG. 3.



Reproduction of photograph taken just before hip-joint amputation.

FIG. 4.

Femur; anterior surface.

FIG. 5.

Femur; posterior surface.

of the body. For this reason, this class of cases requires more radical operative treatment than the other forms. In the common form of necrosis, if the focus of disease is freely opened and curetted, and treated as an open wound in the ordinary manner, the process of repair is speedily established, and in the case of children particularly, the defect becomes filled up with new tissue, and a cure results.

In the diffuse form, however, as in the example here described, the most thorough drainage and antiseptic treatment have no influence whatever in arresting the course of the disease, and there is no tendency to a healthy reparative action. It is therefore necessary, when we become assured that we are dealing with this type, to make a free resection of the bone involved, or to amputate the limb. The decision between these two procedures must be arrived at by a consideration of the probable usefulness of the limb after the operation. If the resection of the entire portion of the bone affected would make a useless limb, an amputation would, of course, become necessary. Failure to remove the disease radically, as has been stated before, usually results in the appearance of foci in some other part of the body. In the present instance, although the amputation seemed to be performed comparatively early, and all the diseased tissues removed as far as could be detected by the eye, there was a recurrence in the stump and evidences of tubercular meningitis developed, from which, apparently, the patient died.

It will not always be possible to detect the diffuse form of osteotuberculosis in the early stages. It should, however, be remembered that in this form the periosteum becomes involved in a plastic exudation, and, therefore, the appearance of swelling about the seat of disease is an early symptom. In the other varieties of osteotuberculosis, the periosteum is rarely involved until later in the course of the trouble.

REPORT OF A CASE OF POST-PARTUM ECLAMPSIA.

By ELIZABETH L. PECK, M.D.

The following case of eclampsia, occurring post-partum, is interesting because of the time intervening between delivery and the convulsive seizure, and also from the fact of accompanying hemiplegia:

E. F., aged twenty-nine years, Irish, admitted to the Philadelphia Hospital, September 8, 1897. She had been delivered at her home, August 29th, of a healthy child. A dispensary physician had attended her during her confinement, and the labor had been normal in every way. The patient had borne four children previous to this child and had one miscarriage. The former labors had been normal. Her health had always been good. The family history, as far as could be obtained, was negative. From the birth of the child, on August 29th, however, she had suffered from severe headache, the pain extending from brow to occiput on the right side. On September 5th, she had four convulsions, during which consciousness was completely lost. The convulsions were preceded by complete loss of power in the left leg and arm.

On admission the patient was dull, complaining only of severe headache. She was said to have passed very little urine in the twenty-four hours previous to admission. The speech was slow and thick. The resident physician found the right pupil slightly more contracted than the left, but this was soon changed. When I saw her, a few hours later, the pupils were equal and reacted well to light. The tongue was protruded straight, and there was no involvement of the facial muscles. The left arm was almost entirely paralyzed. There was ability to slightly flex the fingers. The left leg was entirely powerless. Sensation was not impaired on either side, though the response to any irritation was slow, owing to the dull mental condition. The knee-jerk and the plantar reflex were normal on the right side. On the left the knee-jerk was absent and the plantar reflex impaired. The tongue was heavily coated, but there had been no vomiting.

Vaginal examination showed the uterus high in the pelvis and undergoing normal involution. A moderate laceration of the cervix was present. Some abdominal tenderness could be elicited on deep pressure.

The heart-dulness was normal in extent. The pulse was 80, and the aortic second sound was much exaggerated. The lungs were normal.

The urine, obtained by catheter, three ounces in amount, had a specific gravity of 1030, and albumin constituted one-third the bulk on boiling. The microscope showed epithelial and granular casts, pus-cells and blood.

The patient was placed on a diet of milk and buttermilk. Cathartics and the steam bath were used vigorously. The skin responded nicely, and on the 9th the urine had increased to twelve ounces; besides, several watery stools were voided.

Improvement was slow but steady, though for some time the patient complained bitterly of the pain in her head. By September 23d the albumin had diminished to one-tenth in bulk, though casts and blood were still present.

The patient subsequently left the hospital in a very good condition, having almost completely recovered from her paralysis. The more rapid return of power in the arm was noteworthy, being the opposite of the usual order of recovery of motion.

Not having the case under observation in its early stages, we cannot be sure of the condition previous to the birth of the child. But the persistent headache following immediately after the birth of the child, is evidence of trouble existing at least from that time.

SYPHILIS OF THE NERVOUS SYSTEM.

By CHARLES K. MILLS, M.D.

GENERAL CONSIDERATIONS.

In approaching the subject of syphilis of the nervous system some matters of general interest need to be considered. It is first necessary to have clearly in mind the distinction between the direct and the indirect effects of the syphilitic infection. In all probability, syphilis is due to the action of a living organism, the disease having its specific bacillus, although this has not yet been isolated. It has not infrequently been compared with the exanthemata, and with reason, although in incubation and in its stages it often, perhaps usually, fails to pursue like them a regularly progressive course.

The first great subdivision which clinical experience, as well as pathological study, would incline us to make is into affections due directly to the continuing action of the specific bacillus, and those which are its more or less remote consequences—into Fournier's *syphilitic* and *parasyphilitic* diseases. The parasyphilitic diseases, also called *metasyphilitic*, are affections which are the issue of syphilis, but are not its first and most direct results. "The specific group," says Mickle,[2] "arises from the specific action, processes, and lesions of

[1] For nearly a quarter of a century the writer has been connected with the wards for nervous diseases of the Philadelphia Hospital, which, as years progress, present a field for the study of syphilis of the nervous system scarcely to be surpassed anywhere in the world, and numerous contributions, especially to the clinical history and pathology of this subject, have from time to time been published by members of the neurological staff, as well as by others connected with the medical and surgical departments. It has been my intention in the present paper to prepare a comprehensive contribution on nervous syphilis, based almost exclusively upon the published and unpublished work of my colleagues and myself. After a general consideration of the etiology, pathology, diagnosis, prognosis, and treatment of syphilis as it appears in all portions of the nervous system, I have presented in some detail clinical abstracts, notes of autopsies and microscopical examinations of cases which have been studied in this hospital, arranging these cases with necessary comments under the most recent clinico-pathological classification. In a very few instances in which the hospital has failed to furnish clinical material, recourse has been had to other sources. As a rule, when the cases have been previously published they have been condensed or summarized for the purposes of this article, and credit has been given by references appended to the abstracts. It has not been possible to make the presentation of the subject absolutely full and systematic, but it is hoped that the use which has been made of the material is such as to show the great opportunities afforded by one hospital.

[2] Brain, part i, 1894.

syphilis. The parasyphilitic have origin in syphilis, are products of its action, under its influence, and without it in all probability would not be present, but although thus proceeding originally from syphilis, are not of a syphilitic (specific) nature." Tabes and its congener, paretic dementia, are the great parasyphilitic prototypes; but other acquired degenerative diseases of the nervous system, and some that do not in the usual sense belong to degenerative affections, are included under the head of parasyphilitic or metasyphilitic diseases. Fournier, for instance, puts in this class some forms of neurasthenia and hysteroneurasthenia, special forms of epilepsy, and muscular atrophy. It is not improbable that amyotrophic lateral sclerosis and some other well-known degenerative affections are, at least in not a few instances, syphilitic in remote origin, and therefore should be regarded as belonging to the parasyphilitic class. Whether or not we accept without reserve all the apparently inherited mental and physical conditions and diseases as parasyphilitic to which Fournier would apply this designation, it should be given to some of them, as for instance, to some of the cerebral palsies of children, to some of the forms of imbecility and developmental insanity, and not improbably to some of the muscular dystrophies and atrophies of childhood. In the so-called parasyphilitic affections like tabes, syphilis truly bears a causal relation to the development of the disease, but this is of a different character from that which it holds to the true specific affections of the nervous system. The specific bacillus or the toxines which result from its action disturb and impair the nutrition of the nervous system, and in this sense syphilis acts rather as a predisposing than as a directly exciting cause of disease. Nevertheless, in a very large percentage of cases of degenerative diseases of the type to which reference is now made, the affection would not have developed in the absence of the syphilitic virus or bacillus. The parasyphilitic diseases, while requiring this reference, will not be considered further in the present article, or, if at all, only incidentally, as may be required in the description or elucidation of some point. It happens that true specific lesions and parasyphilitic degeneration are present in the same case, and this combination may require consideration.

From the standpoint of localization, syphilis of the nervous system can best be subdivided into (1) encephalic or cerebral (using the word

"cerebral" here in its broad significance for the entire brain), (2) spinal, (3) cerebrospinal, and (4) peripherospinal (lesions affecting the spinal nerves). As has already been indicated, any one of the subdivisions of the nervous system may be separately affected; any two or more parts may be attacked at the same time, or all may simultaneously be the seat of an active specific disease. The difficulties of subclassification are great. As each phase of the subject is taken up a subclassification will be given. The most practical classification for working purposes is one which considers types or varieties of syphilitic disease under names indicating both the nature and the localization or diffusion of the lesion. When, for instance, a circumscribed syphilitic meningitis of the convexity, a diffuse basal gummatous meningitis, a focal encephalitis, a chronic diffuse meningomyelitis, or a circumscribed softening of the thoracic cord due to syphilitic arterial disease is spoken of, the designation is clear and explicit, which more than counterbalances the objection which may be made to its length. In the present state of our knowledge of this subject therefore the best method of classifying and subclassifying is one which describes the morbid anatomy of the disease in question in terms both topographical and pathological. Fortunately, the symptomatology and pathology of nervous syphilis have now been so well studied that we can confine ourselves to a clinico-pathological classification. The time has come to abolish from our terminology of types and subtypes of nervous syphilis all those terms which simply point to some symptom or syndrome which is presumably due to syphilis, but may be, and in other instances is, dependent upon other forms of lesion. A classification of intracranial syphilis like that of Oppenheim—one that recognizes both the nature and the location of the lesion—is therefore certainly to be preferred to that of Fournier, where the terms used are semeological or clinical (cephalalgic, congestive, epileptic or convulsive, aphasic, mental, paralytic). Such clinical terms standing by themselves tell us nothing of the underlying cause or condition, while an expression like meningitis of the base of the brain, or a gumma of the convexity or of some special convolution, gives us something definite to which to anchor our further consideration of the subject.

The *localization* in the nervous system of specific lesions—in other words, the topographical aspects of the subject under consideration—is of the highest importance. Important also are the facts that true

specific lesions are sometimes disseminated, often diffuse, and still more frequently are multiple, the diverse lesions appearing at successive epochs in the general course of the affection in a particular case. The usefulness or the life of the individual may, on the one hand, be threatened by a lesion or lesions of small size at the base of the brain, or on the other hand, diffuse or somewhat widely-disseminated lesions may cause, at least for a long time, but little disturbance or impairment, either mental or physical. Widely-disseminated syphilitic lesions are usually of moderate size, and they may be minute or microscopical, thus causing special difficulties both in general and topographical diagnosis. Diffuse lesions may spread in continuity over large areas of the cerebrospinal axis, or they may exist as separate large patches in several localities. The multiple lesions of nervous syphilis often give rise to a confusing succession of symptom-pictures, and perhaps eventually to a syndrome of puzzling complexity. In the majority of cases of well-marked nervous syphilis the lesions are sooner or later present, both in the intracranial and the intraspinal portions of the neuraxis; in other words, strictly speaking, pure brain syphilis and pure spinal syphilis are comparatively rare; but while this is true, the dominating symptoms in perhaps two-thirds of the cases are commonly either cerebral or spinal. In a considerable percentage of cases, approaching a third, the existence of well-marked cerebrospinal lesions is evident. Purely neural cases are comparatively rare, although in many forms of cerebral, spinal, and cerebrospinal syphilis lesions of nerve-roots hold an important place.

To say that syphilitic virus is the cause of syphilis of the nervous system is perhaps too much like saying that black is black and white is white, but at the same time it must be remembered that not only may an individual be exposed to syphilitic infection and escape, but he may be infected and yet escape from implication of his nervous system; and more than this, in the absence of exciting and predisposing causes the virus may fail to act, when otherwise it would be efficient. Just why in one the nervous system is more likely to suffer than in another from the effects of syphilis can never be clearly demonstrated. Accurate personal observation, and a truly scientific use of statistics, may help us to a discovery of this reason. The most that we know at present about this matter can be expressed in a few words. Syphilis of the nervous system is more likely to develop in one whose

manner of living and whose hereditary tendencies make this system less resistant to toxic agents. Experience would seem to show that, so far as men are concerned at least, indulgence in excessive venery, so common of course with those affected with syphilis, is a predisposing cause. The neurotic or neuropathic constitution also affords a predisposing soil, although this would seem to be more especially true with regard to parasyphilitic diseases, like tabes. A syphilized neuropathic individual furnishes the easiest victim for parasyphilitic (metasyphilitic) diseases. Those who are predisposed by heredity to vascular and visceral diseases, to arteritis, nephritis, to cardiac or hepatic diseases, are good subjects for the true specific lesions, and are less frequently the victims of parasyphilitic affections.

Several interesting special matters are worthy of consideration in the discussion of the pathology of syphilis of the nervous system, as, for instance, the question of latent or forgotten syphilis and that of the time after infection when the nervous system is most commonly attacked. While in clearly recognizable syphilis of the nervous system a history of exposure is most commonly admitted—perhaps in fully 80 to 90 per cent. of cases—in the smaller percentage it is stoutly, and no doubt in a few instances honestly, denied. This fact is to be explained by the occasional infection by unusual methods, by want of knowledge of the true state of affairs, as when the husband or wife is unacquainted with the existence of syphilis in the other, and by actual loss of memory regarding primary infection. One meets with cases in which the patient is unquestionably innocent of any knowledge that he has had the primary disease. The disease, as Gowers and others show, is sometimes latent or hidden, and the first knowledge of its existence comes when the clinical phenomena of nervous syphilis confront both physician and patient. Wives have been infected with syphilis by husbands who were not aware that they were suffering from the constitutional disorder, and even the reverse of this has, in some instances, been true. Syphilitic offspring are, in rare instances, the first evidences to husband, wife, and physician of the existence of syphilis in either parent. While this is true, such cases are comparatively rare. No statements of much value can be given as to the length of time after infection when syphilis of the nervous system is most likely to occur. A range of variation in time, based upon studies of unquestionable authenticity, is very wide, reaching

from a few months to at least twenty-five or thirty years. The long-held view that the nervous system is very generally attacked at a period after infection to be measured at least by years cannot be successfully combatted, but recent or precocious syphilis of the nervous system is more frequent than was formerly supposed. In my own experience I have seen cases occur within a year, and somewhat frequently within two and three years after infection. Authentic cases in which nervous syphilis has occurred between six months and a year after infection are accumulating. Whether the nervous system of an individual who has once had syphilis is ever absolutely safe may be questioned, and it may be equally questioned whether others are absolutely safe from infection by him. It is simply a matter of taking chances. The chance of escaping is greater according to the time which has elapsed, to the hereditary tendencies of the individual, and to his exposure to those agencies which excite to activity the potential syphilitic virus.

Regarding the etiology of syphilis, Mœller[1] has collected some interesting historical data. In 1610, Gudrinoni found syphilitic gummata in the brain of a man who suffered from headache, epileptic fits, deafness, and somnambulism, and who had been treated with guaiacum. Writers of this period concern themselves principally about the origin and treatment of syphilis. Medicine was, for the most part, speculation, and this continued until the time of Astrue. He describes a host of nervous disorders from syphilis: various forms of headache, giddiness, convulsions, epilepsy, paralysis, and chorea. As causes of these he mentions exostosis, or caries in the bones of the cranium, nodi, or ganglia in the pericranium, abscesses, or gummata in the brain itself, and disturbances of the circulation. He explains such symptoms as giddiness, paralysis, and convulsions as depending on such changes in the blood, lymph, and the tissues as will bring about movements of what he calls *esprits animaux*, which he presumes exist in the lumina of the nerve-trunks.

Mortgagni's investigations led to the discovery of gummata in the cortex of the brain, thickening of the membranes, and their growing together with cortical adhesions. He also found syphilitic processes in the arteries of the brain, as for instance, in a man fifty-nine years of age. DeHorne demonstrates thickenings in the

[1] Nordiskt Med. Arkiv., vol. xxii, No. 22. Abstract in Rev. Insan. and Nerv. Dis., vol. i, p. 37, June 1861, from which the historical data given in this paper are cited.

vessels of the membranes of the brain of three younger syphilitic persons.

Observations like these were however very few. Vigarous, Carrère, Swediaur, and Hufeland referred nearly every chronic disease to the "lues venerea" as a probable cause.

The exaggerations of Astrue's school gave rise to a reaction which was brought about by Hunter. In his work, *A Treatise on the Venereal Disease*, 1776, he says: "It seems as if some organs were less susceptible to the 'lues venerea' than others, and many are, in my experience, not susceptible to it. I have never seen the brain nor the heart, the stomach, the liver, the kidneys, or other viscera affected, though cases are described by writers." His authority in this respect, as in that of the identity of the poison of gonorrhœa and syphilis, prevailed for a long time, so that his ignorance of visceral syphilis served as a proof of its non-existence. This assumption stopped the progress of the syphilitic theory for half a century. A few cases were reported in periodicals in connection with nervous derangements, but they were explained on the old principle that they had their root in the syphilitic products of the surrounding bones. Syphilitic changes in the internal organs were, however, again affirmed by the works of Ricord and his school. He himself demonstrated a case of gummata in the brain to the Académie de Médicin in 1846. On post-mortem examinations of persons afflicted with tertiary syphilis and nervous disturbances, Lallemand, Rayer and others found abscesses and tumors in the brain. It was above all in the mode of origin, transmission, and external phenomena of syphilitic diseases, that rapid and genuine investigations were made in the thirties and forties, but the visceral affections remained obscure to a certain extent. In 1858 Virchow laid the foundation of the anatomical character of the syphilitic products, which is still prevalent. He showed that the anatomical process in the internal and external organs was the same, and that the former were not free from the irritating processes which generally corresponded to syphilis in its early stage. But as to the existence of this disease in the nervous system Virchow had no particular illustrating case to record. During the next decade important works on this subject were, however, published by Oedmanson and Lunggren, and in 1874 Heubner introduced a new factor into the discussion on nervous syphilis which had hitherto been overlooked, viz., the changes in

the walls of the vessels. All the discoveries made in the seventies showed that syphilis was a near or remote cause of disorders in the nervous system, not only in the brain, but also in the spinal cord. It was in reality not until then that the existence of the disease in the latter was acknowledged, though doubtful cases were reported some time before.

In the great works on syphilitic diseases of the nervous system by Lagneau, Zambaco, Le Gros and Lanceraux, published at the beginning of the sixties, are several cases of paraplegia from syphilis.

A step forward was made by Lanceraux in his great work in 1866. He says that syphilitic lesions in the spinal cord differ from those in the brain only in position and less frequency. They may be situated either in the membranes or the substance of the cord. As a proof of the correctness of this view Potain states that in the case of a twin child who died three days after birth, and who suffered from hereditary syphilis, the spinal cord was small, firm, and presented the appearance of a fibrous string of reddish-gray color. Under the microscope no ganglion cells nor distinctive nerve fibres could be discovered. Other writers corroborate the opinion that syphilitic paraplegia may exist without appreciable changes in the spinal cord, whereas, some have found degeneration tumors or hyperæmiæ, with arteries excessively filled. Mere microscopical examinations are thus insufficient to establish a case of syphilis of the spinal cord.

They do not give an absolutely conclusive result any more than do the symptoms of paraplegia, which are principally the same whether due to syphilis or other causes.

Mœller next describes, with considerable minuteness of detail, twenty-four cases gathered from the literature, chiefly giving facts of pathological and anatomical interest, referring only briefly to their history, ignoring altogether numerous cases of syphilitic myelitis reported in connection with them; the disease does not seem to have any *sui generis*.

Mœller found in fifty cases on record where syphilis of the cord had been demonstrated that the time elapsing between the infection and the symptoms of paraplegia was as follows:

In four cases, one-half year; in sixteen cases, between one-half and one year; in ten cases, between one and two years; in four cases, between two and three years; in three cases, between three and five

years; in four cases, between five and eight years; in five cases, between eight and fifteen years; in three cases, between fifteen and eighteen years; in one case, over twenty years.

A few points of general interest in the etiology of hereditary nervous syphilis may here be made, chiefly condensed from Mickle's monograph. Some of the offspring from a man suffering from brain syphilis or any of the forms of syphilis of the nervous system may be the early or late victims of inherited syphilis, the syphilis in this case however not necessarily and perhaps not usually showing itself in disease or arrested development of the nervous system. Disease of bone, joints or skin, malformations of various sort, and disease of almost any viscus may be transmitted by a father who suffers from nervous syphilis. The same is true with regard to the mother. As above stated when referring to parasyphilitic affections of the nervous system, many forms of organic disease or aberration affecting the nervous system are due to syphilis received from a parent, and in these cases nervous syphilis may or may not be present. Sometimes one or several children escape when others fall victims to the disease; sometimes the mother escapes or apparently escapes when the child is affected. The woman may acquire syphilis after the beginning of pregnancy, having both primary sore and that condition of the blood which Mickle speaks of as syphilemia, and in many such cases the child's blood becomes syphilemic with dire subsequent results. When the mother remains apparently healthy, while both father and child are clearly syphilized, it is probable in many cases at least that syphilis is latent in the mother's system, and one proof of this would seem to be the fact that a woman apparently healthy is often not infected by the suckling of a child evidently syphilitic, and sometimes by one who has local syphilitic lesions.

A form of uniformly arrested development of one side of the body, of which I have seen frequent examples both at the Philadelphia Hospital and at the Pennsylvania and the New Jersey training schools for feeble-minded children, is attributed by Mickle to inherited syphilis, and I believe with correctness, although the admission records of these institutions are not always of such a character as to determine the effect statistically. In many of these cases the patients are the victims of epilepsy, which may be more or less unilateral in its manifestations. While hereditary affections of the nervous system due to syphilis more

commonly belong to the parasyphilitic class, true syphilitic specific hereditary disease of the nervous system sometimes occurs. It is clear that this may be the case from the briefest consideration of the fact that the blood of the fœtus as well as that of the mother may, as has been shown, become syphilemic. I have seen a case in which a gumma of the dura, fibroid thickening of the pia, and various anomalies and defects both of brain and spinal cord were present in an epileptic imbecile child dying at the age of twelve. Siemerling[1] reports a case which at the age of four became hemiplegic with aphasia, almost completely recovered, and later became almost blind, was deaf and ataxic. Within a year of the death of the child, vomiting, vertigo, headache, and epileptiform seizures with loss of consciousness were present. Apparently the mental state remained comparatively good, at least until a very late period. The lesions present were gummy encephalitis, basilar gummy arachnitis, gumma of the dura, atrophy of the dura of the convexity, atrophy of the calvaria, multiple osteoporosis of the skull-base, spinal gummy arachnitis.

In the presence of hereditary, as of acquired syphilis, traumatisms are more efficient in the production of hemiplegias, monoplegias, aphasias, and other symptoms and syndromes with a cerebral basis. The train of events most probable usually is syphilemia, some form of syphilitic arteritis, unusual fragility of the vessels, and hemorrhages, small or large, causing destruction of tissue and pressure on neighboring parts.

It must be always borne in mind that syphilis may be acquired even in childhood when considering in a special case the probability of disease being due to inherited syphilis.

Sometimes in a single case of cerebrospinal syphilis, such as occasionally comes to the autopsy-table at the Philadelphia Hospital, every form of syphilitic lesion described as occurring in different so-called types of nervous syphilis may be found on careful macroscopic and microscopic investigation.

Even in a well-marked case of advanced spinal syphilis, all or nearly all the lesions of a nervous syphilis may be detected.

The lesions, gross and microscopical, found in a case which died in the wards of my colleague, Dr. F. X. Dercum, and made the text

[1] Arch. f. Psych., Bd. xx, H. i, p. 101. Cited by Mickle, J.: Brain, part i, p. 135, 1895.

of a paper on spinal syphilis by Dr. William G. Spiller,[1] were for instance of this character. The patient had severe pains in her lower limbs, and in a few days was completely paraplegic, subsequently developing limited anæsthesia, retention of urine, pain in the trunk and along the costal nerve tracks, cystitis and great emaciation.

Macroscopically, Dr. Dercum found adhesions of the dura and extensive fibrino-purulent exudate on both surfaces of the cord, also enlarged and diseased vessels, and some softening of the cord, especially in the lumbar and lower thoracic regions.

Dr. Spiller found that the infiltration within the meninges consisted principally of uninuclear cells, although some few multinuclear were found. In the lumbar region the lesions were of much less intense degree than in the thoracic, and the walls of the vessels were not greatly thickened. In the larger arteries there was only a slight thickening of the intima. A round-cell infiltration of considerable intensity was noticed within the coats of the vessels and the surrounding meningeal tissue. At the level of the first lumbar segment the right posterior spinal root was greatly degenerated, while the left was only slightly affected. The motor cells were highly pigmented; some were tumefied; in some the nucleus was no longer central, and many presented vacuolation of intense degree. By the method of Marchi the posterior columns and the crossed pyramidal tracts were greatly degenerated from the lumbar region upward through the cervical, and the reflex collaterals of the lumbar region showed distinct signs of degeneration. From the lumbothoracic junction to the midthoracic region the lesions acquired more importance, but above this point they gradually became less intense. Some of the cells of the columns of Clarke were merely masses of granular pigment. In the midthoracic region, and especially at about the seventh segment, many miliary gummata were observed within the meninges, and the sclerotic tissues had intimately united the meninges and cord both in the anterolateral and posterior columns. The lesions in the greater part of the cord were more intense on the posterior aspect. The dura and pia were united in the midthoracic region. Small gummata were noted about the walls of many small meningeal vessels, and there was an increase in the number of pial vessels. The posterior roots showed gummatous change, and the few axis-cylinders present in these were

[1] N. Y. Med. Journ., Sept. 25, 1897.

swollen. The round-cell infiltration here was intense, and there was also some about the vessels within the cord. At the level of the first thoracic segment the lesions were comparatively unimportant, and the same is true of the upper cervical region. By the method of Marchi some degenerated fibres were noticed in the pyramids. Endarteritis of the basilar arteries and its branches was present.

The manner in which the parenchyma of the spinal cord is affected in syphilitic myelitis or sclerosis is a much disputed question; and the same questions, of course, are at issue in the discussion of acute or chronic parenchymatous degeneration of the brain substance. Degeneration of the true nerve elements may be due directly or indirectly to vascular disease, although it is also possible that it may in some instances be due to the direct action of a virus on the nerve-cells. Whatever theory be adopted, the true parenchymatous change in spinal syphilis is primarily a degenerative one. Cell bodies and their processes degenerate sooner or later, and the important symptoms presented are due to this fact. It is likely that this degeneration of the noble elements of the central nervous system is most frequently due to lesions of the structures which nourish it; in other words, due to vascular or lymphatic disease. A necrosis of the true nerve elements takes place because the supply of nourishment is in part or altogether cut off by reason of vascular disease. In some cases the inflammation is propagated directly from the meninges, or perhaps from the vessels themselves and interstitial tissues to the parenchymatous tissues. Usually however the destruction of the parenchyma is necrobiotic and secondary to disease of its supplying vessels.

The conditions present in sclerosis of the cord from syphilis are described by Spiller and Sottas as follows:

"Writers speak also of the induration of the cord from sclerosis, and they agree in recognizing that if the softening characterizes the rapid forms the sclerosis pertains to the chronic forms. The sclerosis is only then the last stage of the process—of the inflammation of the interstitial tissue of the cord.

"We would call attention to the fact that the issue of the sclerosis is not always of the same nature. Sometimes—and this is not common—it is a cicatricial tissue resulting from the organization of a specific infiltration. The gummatous infiltration arising from the pia

or the vessels is characterized at first by an embryonic proliferation which can invade, more or less, the medullary parenchyma, and produce later a sclerotic tissue. The sclerosis gives a special appearance; the neuroglia does not enter into its constitution.

"Usually, the sclerosis is purely of the neuroglia; it is preceded by an inflammatory reaction of its own tissue, but this irritation is consecutive to the destruction of the noble elements. In cases of rapid evolution it is possible to constitute the destruction of the nervous elements at a time when the neuroglia does not present a trace of reaction. It is this neuroglia sclerosis which forms the zones of degeneration in the cord. Sometimes it is diffuse, and seems to be the result of a previous state of softening; sometimes it assumes a systematic disposition, and produces the secondary degeneration of the columns of white matter."[1]

Unquestionably, many cases of so-called myelitis from syphilis and other causes are best explained on the theory of ischemic softening. In so-called compression myelitis the softening is due to necrobiosis and is caused by external pressure. The paralyses which follow infectious diseases in general are probably of vascular origin. It has been thought that the anastomoses of vessels were so free in the cord that its ischemic softening was not nearly so likely to occur in the spinal cord as in the brain. The following description of the spinal circulation by Sottas and Spiller shows that the points of distinction between the cephalic and spinal circulation are not so great as was at one time supposed.

"The chief objection to the vascular theory is in regard to the distinction made between the cord and the brain circulations, and to the anastomosis of the cord. These anastomoses only exist for vessels of a certain order; they can also explain a certain number of phenomena, as the collateral congestion and the capillary hemorrhages which accompany the medullary softening. The small vessels are however end arteries, and their injury naturally affects the corresponding parts of the medullary parenchyma. The cord receives its blood supply especially by the arteries of the anterior and posterior roots, branches of the spinal vessels from the vertebral, intercostal, lumbar, and sacral arteries. These branches perforate the dura and divide into an ascending and descending branch which anastomose with corresponding

[1] Internat. Med. Mag., October, 1895.

arteries, and thus are formed arterial chains of which the principal are, an anterior supply, situated in the anterior fissure and formed from the arteries of the anterior roots (tractus arteriosus anterior); then there are two symmetrical posterior chains (tractus arteriosi posterolaterales) situated between the posterior roots and the corresponding lateral columns. From these systems a network in the pia is formed, and from this network the arterioles and capillaries penetrate into the substance of the cord. Once detached from the pia, these small vessels do not anastomose.

"The gray substance and the white substance constitute two separate vascular departments.

"The gray substance is irrigated by the central arteries, which arise from the anterior chain, and according to some writers, there is an anastomosis near the central canal. At different points in the cord these branches penetrate into the gray commissure at the base of the anterior fissure, and having attained one or the other side of the central canal, divide into ascending and descending branches which anastomose with similar ones. The artery at each part of the cord goes only to one side of the central canal—sometimes to the one, sometimes to the other side.

"The white substance is furnished by arterioles from the whole periphery. The anterior columns and the anterior part of the lateral columns are tributaries of the anterior arterial chain, while the posterior columns and the posterior part of the lateral columns, comprising the pyramidal tract, are under the control of the postero-lateral arteries. According to Kadyi, there is a zone of white substance surrounding the gray, which is supplied by both systems. We have observed that the nervous tissue of this zone was always more respected, even when the peripheral and central parts of the cord were very much altered.

"It is necessary to examine the condition of these peripheral end arteries; often they are much more altered than are the vessels from which they arise. The vascular systems may be involved separately, or at least the lesions can predominate in one, and thus give rise to the appearances of systematization which medullary syphilis often assumes; in this way can be explained the poliomyelitis anterior of rapid evolution in a syphilitic subject. We

would expect the vascular changes to predominate in the anterior system in such a case.

"The veins of the cord anastomose freely everywhere. The largest of these are situated at the posterior and anterior median fissures. In the syphilis of the cord the veins are usually more affected than the arteries, and these lesions, in spite of the anastomosis, have an important bearing."[1]

In the softening which results from encephalitis or myelitis, more or less complete destruction of the nerve-cells, bodies and processes takes place. The writers cited above describe the appearance present in softening of the cord due to syphilis as follows.

"The intensity of the softening varies in degree. There may be complete destruction of the nerve tissues and the formation of a creamy substance. The process consists in a destruction more or less advanced of the noble elements with the formation of the products of necrosis, amorphous, granular, albuminous substance, granules of myelin, granular bodies, etc., and in a special alteration of the interstitial tissue peculiar to the nervous parenchyma. There is an exaggerated development of Deiter's cells. These modifications are not special to syphilis. In these cases there does not exist a trace of inflammation of the interstitial tissue, which proves that the destruction of the noble elements is not consecutive to an inflammation of the interstitial tissues, and that it is necessary to seek another explanation for this phenomenon."

In cases of hereditary syphilis of the nervous system the lesions found are the same or similar to those noted as present in the acquired disease. Comparing equal numbers of cases due to heredity, and those acquired, the lesions will probably be found of less intensity in the majority of the former, although they exist in equal grade in a minority of cases. The lesions may be of bones, membranes, vessels, and parenchyma; the osseous lesions may be thickenings or enlargements of the bones, and caries and periosteal nodes may be present. Both internal and external pachymeningitis have been observed in cases of inherited syphilis. Thick, fleshy, vascular membranes have been found covering the internal surface of the dura, comparable in nearly all respects to the neomembrane of internal pachymeningitis due to acquired syphilis and other causes. Lymphatics, capillaries,

[1] Internat. Med. Mag., October, 1895.

veins and arteries have all been the seats of inflammation, endarteritis being especially common. Other recorded lesions are dural and pial gummata, gummatous leptomeningitis, focal and diffuse encephalitis and myelitis, necrobiosis from obliterative disease of arteries and veins, and fibroid indurations, usually secondary to chronic vascular disease.

In basal gummatous meningitis the macroscopic appearance of the membranes may vary from a mere opacity with slight thickening to a diffuse gelatinous mass; the arteries are changed in calibre, the walls thickened and their lumen sometimes obliterated, the nerves are discolored and irregularly swollen. Necrosed and hemorrhagic areas are found in varying number.

"Histologically, we find the new formation to be composed of granulation tissue, rich in cells and vessels and containing fibrous and caseous foci. Circumscribed gumma is rare. The arteries are always involved. The entire vascular wall is thickened by a small-celled infiltration. Infiltrations into the intima contract the lumen of the vessels. The nerves show microscopically that their degeneration is dependent upon infiltrations which radiate from the epineurium between the fibres. These are dense fasciculi of fibrous tissue containing small cells and numerous vessels. While the nerve-trunks enlarge the fibres atrophy, the cerebral substance suffers by the rupture and obliteration of its vessels."[1] (Oppenheim.)

In gummatous meningitis of the convexity the lesions are similar in kind to those found at the base, but perhaps are usually less intense. Old or recent gummata, opacities and gelatinous exudations, thickened, obliterated or broken vessels, with areas of softening or of hemorrhage, are the common lesions.

The basilar artery shows a particular tendency to become enlarged and thickened, but the same tendency is shown by other great vessels at the base, and especially the middle cerebrals. The lumens of the small branches of these vessels are frequently closed, causing interior softenings and resulting monoplegias, hemiplegias, aphasias and other symptoms or syndromes indicative of destructive focal lesions.

In spinal syphilis the lesions are osseous, membranous, vascular, or parenchematous. In the osseous and meningeal cases they may be more or less limited, appearing as caries, periosteal thickenings, external and internal pachymeningitis localized or diffused,

[1] Neurolog. Centralbl., No. 15, 1890.

and sometimes as leptomeningitis, although almost invariably when leptomeningitis is present the cord is to a greater or less extent involved, giving a meningomyelitis of a moderate or a severe grade.

In the diagnosis of spinal syphilis and also of some forms of intracranial syphilis, especially basal meningitis, the consideration of a few points will be of great value. The pathological condition produced by the syphilitic lesion is in most cases a meningitis which is associated in greater or less degree with a myelitis or encephalitis. Necessarily, owing to the localizations of such lesions, the spinal or intracranial nerve-roots frequently become involved, and it is also frequent, as just indicated, to have invasion of the nervous parenchema. What would be the nature of the symptoms resulting from lesions of the character and in the localities indicated? They will, in the first place, be symptoms of irritation—pains due to root-neuritis or at least to irritation of root-fibres; spasmodic symptoms such as clonic or tonic spasm, the latter including rigidity of the back in spinal cases, and various forms of paræsthesia. Secondly, they will be symptoms indicative of pressure, these including both cord and brain symptoms and phenomena due to compression of nerve-roots—paresis or paralysis; anesthesias—interference with reflex action, vasomotor and trophic symptoms, etc. Thirdly, the symptoms will indicate absolute destruction of the substance of the cord or of the brain—these being much the same as in the second class but more prominent, namely, paresis or paralysis, anæsthesia, etc.

In this connection a reference to a recent contribution of Oppenheim on the so-called Brown-Séquard type of paralysis is interesting.

According to Oppenheim[1] nearly one-half or more than one-third of the cases of Brown-Séquard paralysis which are usually somewhat impure or irregular in type, are due to spinal or to cerebrospinal syphilis. In this contribution he refers to a few of the phenomena in the cases coming under his own observation, these having received comparatively little attention.

They are phenomena of irritation, which in one case preceded the development of the Brown-Séquard symptom-complex, in the other appeared in the course of the disease or after the termination of the trouble which had produced it. They appeared simultaneously in the

[1] Centralblatt f. die medicinischen Wissenschaften, No. 12, March 25, 1899.

motor and sensory systems, and consisted of increased tonicity of the muscles on the side of the lesion and pain in the opposite half of the body. In a case in which the lesion was situated in the upper cervical cord of the left side, and in which spastic paresis existed in the left extremities and anæsthesia in the right, tonic spasms were observed occasionally in the left arm and leg, simultaneously with violent pains in the limbs of the right side of the body. Usually pain was also felt in the distribution of the left upper cervical nerves. This *spasmodinia cruciatia* (tonic spasms on one side, pain in the contra-lateral side of the body), as Oppenheim suggests one might possibly call these phenomena, resembles very closely the Brown-Séquard type. He reports another interesting case, which he believes was best explained on the theory of disseminated sclerosis, although it seems to the writer it might have been equally well explained on the assumption of multiple or disseminated syphilitic lesions.

It is an error to always conclude that because an individual has a history of exposure or even of infection, that symptoms or diseases which might be referred to syphilitic lesions are dependent upon syphilis. It will be necessary to refer to only one or two illustrations of this point. In several cases of brain tumor, the lesion either through autopsy or operation proving to be sarcomatous, the patient has had a history of syphilis. In other cases with a history of syphilis, nephritis with the often accompanying disease of heart and vessels has been present, the disease of the vessels resulting in necrosis and degeneration of brain tissue. In such cases antisyphilitic treatment is often pursued with persistence, and sometimes to the injury of the patient. It is equally true, on the other hand, that syphilis cannot be excluded because of the absence of a syphilitic history, and this even in cases where one has no reason to doubt the truthfulness of the patient; in other words, a patient may have been infected and not know it. Every practitioner of large experience has seen not a few such cases. The simple inference from these facts is that a diagnosis should never be based simply on history or the absence of history, although the importance of either should not be discredited.

The most important features in the general diagnosis of brain syphilis are well presented in the following quotation from Oppenheim: "Tubercular meningitis is to be excluded by reason of its development, course, and temperature. In syphilitic meningitis the temperature is

normal or is only occasionally increased; but the most important differential point is the peculiar course of brain syphilis, the *variability of the symptoms*, their *coming and going and changing* constantly. The visual disorders may also undergo this *oscillation*, sometimes to an astonishing degree. I found, for instance, in several cases under examination on various days, now a normal visual field, now an irregularly concentric contraction, whose borders varied from day to day, now a pronounced hemianopsia, etc. A *fugitive bitemporal hemianopsia* seemed to me to be particularly characteristic. A temporary and remittent amaurosis, also a remittent choked disc may occur. The same is true of the paralyses of the ocular muscles. To-day we find a paralysis of the oculomotor; after a few days it may have markedly receded, to return shortly in its full intensity and completeness. I treated a patient in whom ptosis and paralysis of the superior rectus appeared repeatedly in the one eye while I was examining her, and which disappeared in ten minutes to half an hour again while I was still examining her. A coming and going of *reflex iridoplegia* was even noticed in one case. The facial paralysis may repeatedly remit, and, as I have noticed several times, may jump from one side to the other. This active variation in the phenomena is explained by the pathologico-anatomic alterations. This granulation tissue proliferates and dies in rapid succession and constant repetition, and the nerve which is surrounded by it is subjected to a more variable pressure than in any other disease. The symptoms described above are caused by a meningitis and a neuritis or gummatous perineuritis of the cranial nerves."

In no branch of medicine and surgery is accurate and thorough diagnostic study of cases more important than when the physician is confronted by the numerous problems of prognosis and treatment in nervous syphilis. About no other subject is so much complacency and optimism exhibited by practitioners young and old. Most men prefer to travel easy roads, and the magic of a syphilitic history solves for them at once the often troublesome problems of diagnosis, prognosis, and therapeutics; and if by chance any doubts there be as to syphilis, while the possibility of it remains they have that happy resort—the therapeutic test. After all, the application of the therapeutic test, either for the purposes of diagnosis or prognosis, is an unscientific method of trying to reach a conclusion, and this is true in spite of the fact that it sometimes is practically useful. One should

be competent to make both a diagnosis and prognosis without therapeutic experimentation, otherwise he is not competent at all for the work in hand. The therapeutic test sometimes leads to delusive opinions and inferences, as when it has been held, for instance, that because tabes fails to yield satisfactory results from antisyphilitic treatment it is therefore frequently not of syphilitic origin.

Many practitioners seem to hail the diagnosis of nervous syphilis with a sense of satisfaction that has underlying it a feeling that in all such cases the prognosis is good. This is far from the truth, although I am not prepared to take the pessimistic ground held by Gowers,[1] that no case of syphilis ever has been or can be cured. It must be noted here however that the real views expressed by this neurologist are sometimes not understood. He does not mean that the manifestations of syphilis can never be removed, nor removed for a time, but that the morbific agency remains in the system. It is possible to remedy some of its effects, to remove some of its symptoms; it is even possible that a cure may be attained, but this has never been proved. He compares syphilis with the exanthematous fevers, which he holds are not influenced by treatment, or at least not cured by treatment, or if at all only in the sense that the symptoms are relieved or removed. This is a very different thing from curing a disease.

For practical purposes cases of syphilis of the nervous system, as of some other more or less intractable diseases, may be regarded as cured when the manifestations of the disease are subdued by treatment for several years, and when the children of syphilized subjects are born healthy and remain so. Cases can be regarded as improved or relieved when the painful, distressing, or crippling symptoms of the disease are removed in whole or in part, and when the patient is restored to partial or it may be nearly complete usefulness, so far as his business capacity and his domestic and social relations are concerned. Looking at the subject in the way in which it appeals to the everyday physician, whether specialist or general practitioner, cases of syphilis of the nervous system from the standpoint of prognosis can be somewhat roughly and yet usefully subdivided into about four classes, and in attempting this classification the effort is based almost entirely upon personal experience in the Philadelphia Hospital and in private practice, although doubtless this experience corresponds with the

[1] "The Lettsonian Lectures on Syphilis and the Nervous System," Brit. Med. Journ., No. 1, 1889.

experience and published work of others. Remember that I am speaking here exclusively of the cases with frank or true specific lesions, the cases of syphilitic disease proper of the nervous system, not the metasyphilitic or parasyphilitic affections. The first class of cases is one in which the diseases present are curable, using the word curable in the sense above explained. This class includes perhaps 15 per cent. of all the cases which come under observation in a large neurological practice, private and hospital. This percentage of cures cannot however be affected unless the cases come under observation within a reasonable time after the symptoms which indicate nervous syphilis have been presented. By this is meant a period of from one to six months, or of a year at the most. Unless the cases come under observation as early as here indicated, the number of absolute cures will probably not exceed 5 per cent. of the number treated. The second class includes about 30 to 40 per cent. of the cases which are left after the withdrawal of the percentage of entirely curable cases. It includes all those cases which can be markedly relieved or improved by appropriate antisyphilitic treatment. The cases here referred to are not only relieved of some of their most distressing and disabling symptoms, but remain relieved for many years, and it may be until the ends of their lives. It will be seen that to the physician, and above all to the patient the ability to differentiate these cases and separate them for the purposes of prognosis and treatment is of supreme importance. In a third set of cases relief or improvement is not infrequently obtained, but it is of brief duration, symptoms which have disappeared under treatment recurring, or new signs and symptoms coming into view. These cases relapse again and again in spite of treatment, and it may be treatment which is apparently of the most appropriate character. In these cases a truce may be made with syphilis, but not a definitive treaty of peace. In a fourth and unfortunately a comparatively large class of cases, syphilitic disease of the nervous system, once initiated, has a steadily downward course. The individual case may last a year or six years, or it may be a month or six months, or its course may be measured even in a proportionate number of weeks or days in extremely rare instances, but this march, whether rapid or slow, is always toward a fatal termination.

With others who have written on this subject, I believe that syphilis as it now shows itself in Philadelphia is milder in its manifestations

than in my early days—say twenty-five to thirty years ago. This is perhaps especially true of the disease as it is seen in lesions of the skin, bone, joints, and viscera of the body. Musser[1] has called attention to this point in a paper published in 1894. As the statistics on which he relies are based on studies in the Philadelphia Hospital, it may be worth while to repeat them here.

Percentage of syphilis in hospital for periods of five years, 1864 to 1893, inclusive:

1864 to 1868—6.68 per cent.	1879 to 1883—4.26 per cent.
1869 to 1873—4.56 "	1884 to 1888—3.76 "
1874 to 1878—3.36 "	1889 to 1893—3.05 "

It is probable that these statistics do not properly indicate the data which should be furnished by the nervous wards of this hospital owing to the differing manner in which the cases have been tabulated for report purposes. The question of the milder type and less frequent occurrence of syphilis needs, as regards the nervous system, to be separately considered. The majority of male adults in the nervous wards have at one or another time been the victims of syphilis (?), while a less, although a large, percentage in the women's wards have also been the subjects of this disease. So far as syphilitic affections of the nervous system are concerned, my own belief is that the syphilitic affections of the nervous system proper have decreased and are of somewhat milder type, while the parasyphilitic affections have increased in number and are less rapidly fatal. This would seem to point either to less virulence in the syphilitic virus or to improved resisting powers on the part of the individual, or both.

By my colleagues of the neurological staff of the Philadelphia Hospital and by myself, both the iodides and mercury are used in the *treatment* of the numerous cases of syphilis of the nervous system which are always to be found in the wards for nervous diseases and in the insane department of this hospital. On the whole, the iodides are used more than mercury, at least by the writer.

The following citation from my paper on "Some Phases of Syphilis of the Brain,"[2] which embodies the substance of lectures delivered at the Philadelphia Hospital, will serve to present my views and practice.

Gowers holds that on the whole the iodide is the most useful and

[1] Medical News, Aug. 11, 1894.
[2] Medical News, 1895.

the most certain of the two drugs; still either may be used with success in most cases. When the iodides fail, which is very rarely if success can be obtained at all, mercury may be used successfully, even for the late manifestations.

The administration of large doses of the iodides, as much as from 400 to 800 grains in a day, has been called the American method. The iodides should always be given in an efficient manner. My usual plan is to begin with doses of from 15 to 20 grains three times daily, and increase by five or ten grains daily until as much as a drachm, or even more, is taken three times daily. I have seldom found it necessary to administer more than half an ounce in a day. On the whole, the amount which has proved most successful is from 2 to 3 drachms daily. If iodism is produced, it may be necessary to discontinue the use of the drug for a time or to diminish the dose, although, strange to say, occasionally when iodism results from the use of small doses it may be made to disappear by rapidly increasing the amount ingested. Undoubtedly in some cases from 300 to 400 grains of potassium iodide or of sodium iodide daily will be well borne and will produce rapidly beneficial results.

Of the preparations of mercury used by the mouth, calomel and the biniodide are to be preferred, the former in doses of from one-sixth to one-fourth of a grain every two or three hours, giving at the same time, if necessary, preparations of opium, such as paregoric or even morphine, in order to prevent looseness of the bowels. The biniodide may be used in doses of from one-twelfth to one-sixth of a grain every two hours, administering after each dose, if the bowels are affected, small doses of paregoric.

The use of mercury by inunction, if this system can be systematically and thoroughly pursued, is one of the best mercurial methods in nervous syphilis. The official ointment of mercury and mercury oleate are the preferable preparations. From one-half to one drachm can be used daily. In order to be exact as to the amount used, a good plan is to divide an ounce of the ointment into one-half drachm portions, wrapping each of these in paraffin paper. Mercurial inunctions and the use of potassium and sodium iodides may often be advantageously combined.

In Germany in particular, and to some extent in this country, under the influence of the teachings of Wolff and others, mercury has

been employed hypodermically, and in some instances with striking success. The insoluble compounds of mercury, and especially calomel, are to be preferred.

Gowers wisely suggests that every syphilitic subject should for five years after the date of his last symptoms have a three weeks' course of treatment twice a year, during which time he should take from twenty to thirty grains of iodide daily. It is better that this rule should be adopted three times during the year, instead of twice.

The treatment can be given in a word or two. When these cases are met in the acute stage, first clear them up, if they have been on a debauch, by purging them and keeping them quiet. Then if the conditions remain, attack them vigorously with antisyphilitic treatment. Take one, two, or three drachms of mercury put up in wax papers, and order these to be used in one day by inunction. Get the effect as early as possible and thus dissipate the early conditions and save the patient from a more lamentable fate. If the patient improves keep up the mixed treatment with iodide and mercury for a time, and after awhile use only an iodide treatment. The rule is much mercury at first, then mixed treatment, later iodide alone—twenty to thirty or forty grains taken at a dose several times a day.

The following from another paper[1] by the author are some general conclusions regarding the prognosis and treatment of syphilis of the nervous system.

The prognosis and treatment of syphilis of the nervous system must always be based on a thorough study of differential diagnosis, the treatment requiring in addition a careful investigation of special forms and of individual cases, the latter including the past history of treatment and a study of idiosyncrasies. In considering both prognosis and treatment it is of the first importance to clearly appreciate the distinction between diseases with specific lesions and parasyphilitic or metasyphilitic affections; the former are improvable or approximately curable, the latter cannot be cured and are as likely to be harmed or not benefitted by the usual and unusual therapeutic procedures adopted because of a history of syphilis. The most improvable and curable of syphilitic diseases are those with recent specific lesions, such as young gummata, recent osseous disease, recent meningitis, recent encephalitis or myelitic infiltration, recent perineuritis, and

[1] Phila. Monthly Med. Journ., vol. i, Feb., 1899.

recently-developed arterial disease—all these, be it noted, being lesions which do not primarily implicate the true nerve elements. In proportion to their destructive involvement the prognosis will be unfavorable and treatment unavailing.

In the discussion of prognosis and treatment practical good results from a consideration of the method of onset of the improvable and curable types of nervous syphilis. Cases of subacute development are relatively most hopeful; acute cases may be rapidly fatal or destructive, but can be benefitted by early recognition, followed by prompt and energetic treatment; cases slowly developed and chronic in course, best illustrated by Erb's syphilitic spinal paralysis, and the most common type of chronic pseudo-paresis, can often be helped but are never cured by antisyphilitic or other treatment, the reason being that in these cases associated with meningeal, myelitic and vessel disease are resultant necroses, indurations, and secondary degenerations. Specific lesions of the nervous system occurring within a year after primary infection are often rapidly fatal, or at least rapidly destructive, but at times their course can be stayed by very early and very energetic treatment.

SYPHILIS OF THE SPINAL CORD AND ITS ENVELOPES.

Syphilis of the spinal cord and its envelopes can be conveniently discussed under the following heads: (1) Syphilitic vertebral disease; (2) syphilitic pachymeningitis; (3) acute syphilitic myelitis; (4) chronic syphilitic myelitis or meningomyelitis; (5) syphilitic tumors of the cord and its membranes; (6) circumscribed softening due to syphilitic disease of the vessels.

Pachymeningitis, whether acute or chronic, when of the internal variety, is frequently, perhaps usually, associated with leptomeningitis, the membranes being adherent. In acute syphilitic myelitis, which is sometimes classified as paraplegia of acute onset, while meningitis may be present it is commonly of slight degree and may be entirely absent. In chronic myelitis meningitis is more commonly present, but in the type under consideration it is of varying degree and usually slight. Syphilitic meningomyelitis is a very distinct affection, and in its most marked type both meningitis and myelitis may be well represented. In all forms of myelitis and meningomyelitis softenings from necrosis and secondary degenerations

are usual, and may be extensive. Syphilitic spinal tumors are as a rule meningeal in origin, but may in rare cases like gummata of the brain originate in the vessel-walls. In any form of leptomeningitis, meningomyelitis or myelitis, vascular occlusion may occur, but in the cases to which reference is made under the sixth head of the above classification, an isolated focus or isolated foci of softening due to closure of particular vessels are understood.

SYPHILITIC VERTEBRAL AND DURAL DISEASE.

Syphilis may attack the bones of the spinal column as it is well known to attack cranial bones. This disease sometimes occurs as an isolated affection, or the lesion may be one of several attacking bone, membranes, and even the cord. Occasionally disease of the spinal bones is consecutive to disease of the cranial bones, and occa sionally also disease of the pharynx becomes by spreading disease of the cervical vertebræ. Mickle[1] believes that syphilis sometimes determines the tubercular disease of the vertebral bones, but he also believes that we may have a purely syphilitic caries. With both of these positions I am in accord, and I have had illustrations of both forms of disease.

Syphilitic growths may be found in the spinal dura. Sometimes these are very numerous, almost miliary in character; again they may exist in several aggregations, and again a gumma of considerable size may be found in one position. Such a gumma may give rise to a special syndrome, as to that well known as Brown-Séquard's paralysis, when the lesion is unilateral; or it may cause a cervical paraplegia, or a paralysis of both lower extremities with symptoms of compression, much like those which are given by a case of tubercular caries or perhaps by a case of hypertrophic pachymeningitis.

Two cases observed by me in the Philadelphia Hospital were reported many years ago[2] at considerable length, and their history somewhat condensed will be given here.

These cases were published under the title of "Syphilomata of the Cervical Dura Mater," and were good illustrations of conjoint osseous and membranous disease in the cervical region. Bone and membrane were involved in inflammatory and destructive disease, and eventually the cord at the neck was compressed.

[1] Brain, part i, 1895.
[2] Phila. Med. Times. Nov. 8, 1879.

W. H., aged twenty-four years; colored; married; for several years had been addicted to excessive venery, and twelve months before coming under observation had contracted a hard chancre. Two months after this he began to suffer with severe pain in the back of his neck, which soon became stiff and slightly twisted. These symptoms of irritation grew worse, and soon were added weakness, first of the arms and later of the legs, with numbness and unpleasant sensations in the limbs. In six months he was bedridden.

On admission to the hospital, two months before his death, he was in a state of pitiful helplessness, bound hand and foot by disease. He was only able to sit up for a short time when fully supported. His neck was very rigid, and seemed to be shortened and shrunken together. His head was held slightly bowed forward and twisted toward the right, any movement of it causing him great pain. He often felt as if he was being choked. He complained of dimness of vision. Both arms were much wasted, the legs however showing but little atrophy. He could not lift either hand from the bed, and could only bend the lower limbs slightly at the knees. The left side was possibly a little bit less helpless than the right. Electro-contractility was preserved. Sensation was diminished in both upper and lower extremities, but was not mapped out. The slightest irritation of the skin would cause his legs to jerk violently. The thighs, apparently spontaneously, would sometimes assume a semi-flexed position to the pelvis, from which he had no power to release them; they would have to be straightened by the attendants. His evacuations were involuntary, and incomplete priapism sometimes occurred. His sufferings could only be palliated. He continued to grow worse. Every night, and often during the day, he was attacked with severe pains in his knees and ankles. No acute bedsores made their appearance, but several small ones, from pressure, formed at different points. On a number of occasions he had most distressing paroxysms. He would all at once be seized with a sense of choking and terrible dyspnœa, and would have to be propped and held up in bed; at the same time profuse perspiration would break out. He finally died in one of these attacks. His average temperature, taken in the right axilla, was for the two weeks: morning, 97.9° F.; evening, 98.3° F.

On cutting through the vertebral column a quantity of pus, and of pus mixed with blood, escaped from the spinal canal, particularly from the right side at the level of the third and fourth cervical vertebræ. The inner faces of the anterior segments of the second, third, and fourth cervical vertebræ were eroded and partly carious, the intervertebral cartilages being also partly destroyed by disease.

On the external surface of the dura, for a distance of two and one-half inches in the upper and middle cervical region, was an exudation or growth, with an uneven, irregular surface, from one-fourth to one-third of an inch in greatest thickness; it was distributed so as to girdle the cord, but was most developed in front. The inner surface of the membrane was smooth, and the cord within the limits of the diseased dura was flattened. The cervical nerves passing through the membranes were compressed and atrophied.

Nearly in the centre of the anterior aspect of the cord, about the level of the third cervical vertebra, was an oval or bean-shaped spot of bulging, half an inch long and one-eighth of an inch wide. It seemed to be defined from the surrounding tissue, looking like a small intramedullary tumor. The cord above and below it was softer than elsewhere. Three inches above the cauda equina was an exactly similar but smaller eminence.

A dural growth and recent hemorrhage were present in the second case, but the vertebræ were only slightly involved.

H. J., aged thirty-one years; colored; married; was admitted to the Philadelphia Hospital August 26, 1879. The history obtained from herself and friends was that in the preceding May she had been taken with a feeling of stiffness and pain in the neck. The pain radiated upward to the head, and to a less extent down her back and arms; it was greatly aggravated by any kind of jarring or jolting; she suffered so much in this way that she soon became unable to ride in the cars. She lost flesh and became weak, but did not become distinctly paralyzed until two weeks before she was brought to the hospital. At this time, while attempting to dress herself, both arms suddenly fell powerless into her lap. The next day she could not stand, and since she had been completely helpless from the neck downward.

On examining her it was found that she was compelled to lie constantly in one position on her back. She could not sit up at all, even with the amplest support. Her head and neck were held perfectly rigid, so that she rested on the right posterior portion of her head. Any attempt to change this position caused extreme pain, which shot upward to the head. Both arms were entirely helpless. She was able to pull up the right leg so as slighty to flex it at the knee, but she could not do this with the left. She could perform movements with her feet, but with diminished facility. Roughly tested with the æsthesiometer, sensation seemed to be pretty good, but her answers in regard to the distance apart of the compass points were uncertain and confusing. Reflex twitchings of the legs were produced when the points were brought in contact with the skin. The knee-jerks were present on both sides, but not exaggerated; the response on the right side was poorer than on the left, the latter being about what is usually found in health. When first examined, on the day of admission, she had full control of her bladder and bowels. She had been troubled more or less for several weeks with shortness of breath, and three days before coming under observation she had had a severe paroxysm of respiratory difficulty in which she thought she was "dying away." The attack was accompanied by profuse perspiration. Her pulse, recorded on the 26th, was 84, weak, but not intermittent.

Treatment was instituted, but without hope, as the patient was when admitted evidently in the last stages of slow compression of the cervical cord. Her bed and pillows were so arranged as to give her head and body as complete rest as possible, and among the other measures adopted were leeches to the nape of the neck, potassium bromide and iodide in combination internally, hypodermic injections for the relief of pain, an ointment of belladonna, iodine, and mercury locally, and belladonna, digitalis, and ammonia for the paroxysms of dyspnœa and exhaustion. Fluid and semi-solid food were also frequently given. She grew worse from day to day; she was often bathed in profuse perspiration; dyspnœa and cardiac irregularity and weakness became greater, and she died, exhausted, August 31, five days after admission. Her temperatures, taken on the evening before her death and on the day of her death, were as follows:

	Right axilla.	Left axilla.
August 30	100 ° F.	102.2° F.
August 31	99.4° F.	99 ° F.

A post-mortem examination was made twenty-three hours after death. Post-

mortem rigidity had not taken place to any marked extent, except in the fingers. No decided wasting of the body or limbs was present. The muscles of the neck were completely relaxed.

No evidence of disease outside of the vertebral column existed. The spinal cord and membranes were examined in position after sawing through and removing the posterior portion of the vertebral column. On the left side of the cord, outside the dura, a layer of dark, clotted blood could be seen, reaching upward from the fifth cervical vertebra nearly to the upper limit of the cord. Externally, the upper portion of the dura for a distance of more than two inches was the seat of an irregularly-flattened exudation or growth, which was found on removal of the cord and membranes to be thickest to the left, in front and above. In the upper part of this diseased mass, at about the level of the second cervical vertebra, was a small, dark clot, about the size of a half-dime. The inner surface of the dura was smooth, but more opaque than usual. After removing the cord and membranes the cervical and dorsal vertebræ were examined minutely, but nothing abnormal was found, except a very slight roughening of the adjacent borders of the inner surfaces of the anterior segments of the second and third vertebral bones.

The upper cut end of the cord was softened and yellowish-white in color. The cord in the upper cervical region was more flattened than usual anteriorly.

The brain and its membranes were examined. The pia showed signs of recent hyperæmia, particularly about the junction of the ascending parietal convolution with the inferior parietal lobule. No other organs were examined.

A microscopical examination of the thickening of the external dura, made by Dr. H. F. Formad, showed it also to be a syphiloma, similar in structure to the specimen from the first case. Sections from the cord below showed likewise a transverse sclerosis.

In the PHILADELPHIA HOSPITAL REPORTS, Volume III, 1896, Dr. Charles A. Vandervoort, resident physician, reported from my service at the Philadelphia Hospital a case studied by me with the reporter, of syphilitic disease of the cervical vertebræ with spinal hemorrhage, the case showing interesting localizing phenomena.

The patient was a colored man, aged thirty years. The autopsy showed the disc between the fourth and fifth cervical vertebræ to be soft and spongy, lacking the dural lining of the canal, and being the seat of old disease, probably syphilitic. The diseased area was nearly the size of a nickle, and presented an appearance altogether unlike that of any other portion of the canal. The spinal cord was found full of dark fluid blood as high as the junction of the fourth and fifth cervical vertebræ. A small subdural ecchymosis was found at the anterior surface of the cord about opposite the origin of the seventh cervical nerve. This man had had a sudden attack which left him completely paralyzed in both legs and his right arm, while the left arm showed a degree of paralysis and anesthesia which indicated the height of the

hemorrhage on that side as about the fifth cervical segment. The case as regards its symptomatology had many interesting localizing phenomena, for which the reader is referred to a previous volume of the REPORTS. Most of the symptoms presented by the patient were due to the intraspinal hemorrhages, but the disease which led to this hemorrhage was chronic and involved the bones and intervertebral spaces. One of the sources of danger in both syphilitic and tubercular caries is sudden hemorrhage.

I have no notes from my Philadelphia Hospital records of cases of typical Charcot's cervical hypertrophic pachymeningitis. In one case supposed by me during the patient's life to be, in part at least, an example of localized hemorrhagic myelitis, the autopsy showed the presence of a unilateral localized pachymeningitis situated about the junction of the cervical and thoracic regions of the cord. The patient had a peripheral type of paralysis in the forearm, with marked atrophy of the extensors and some other groups of muscles, and some irritative sensory symptoms. He had other symptoms, both spinal and intracranial. Some of these were pressure effects, and others were due to multiple syphilitic lesions. He had a syphilitic history.

ACUTE SYPHILITIC MYELITIS.

Under the name of acute syphilitic paraplegia, syphilitic paraplegia of acute onset, acute syphilitic spinal paralysis, etc., an interesting class of cases has been well described by Schmaus,[1] Sottas,[2] Sottas and Lancereaux,[3] Goldflam,[4] Rosin,[5] Möller,[6] Williamson,[7] and others. I have seen numerous illustrations of this type of syphilitic spinal disease during my long service at the Philadelphia Hospital, and a few cases have come under my observation outside of the hospital. These cases usually reach their maximum severity in a few hours or in a few days at least, although they may have a prodromal period of considerable length. Sometimes even the prodromal period is short. The cases as observed by me are of at least three varieties as regards severity: (1) a variety relatively rare, in which the disease passes rapidly to a

[1] Deutsches Arch. f. klin. Med., Bd. xliv, 1889.
[2] Paralysies Spinales Syphilitiques, Paris, 1894.
[3] Paralysies Spinales Syphilitiques, Paris, 1894
[4] Wiener Klinik, p. 65, 1893.
[5] Zeitschr. f. klin. Med., Bd. xxv, 1896.
[6] Archiv. f. Dermat. u. Syphilis, 1891.
[7] Syphilitic Diseases of the Spinal Cord, Manchester, 1899.

fatal termination; (2) a common variety, in which the symptoms are severe at first, but slowly improve or subside and the patient makes a partial recovery, the degree of restoration varying in wide limits; and (3) a mild form, in which the patient, at first attacked with greater or less severity, makes a nearly complete recovery.

E. H., aged thirty-five years; white; domestic; married; was admitted to the hospital August 23, 1895.

The patient stated that she had been well and able to work until attacked in July, 1900. On admission she had pain in a circle around the body. It hurt her in the back to walk; four or five days afterward she began to lose power in the leg. She had to be catheterized, as she could not pass water; her bowels were incontinent.

She had a history of chancre ten years ago, followed by secondary eruptions. Paralysis came on rapidly, becoming complete in about four days. The left side was involved first, the right a day or two later. Loss of power in both legs and in the abdominal muscles was complete. She had girdle pains for two days before the paralysis, which pain gradually disappeared with the development of the paralysis.

The following notes were made about the time of admission: Muscle-jerk and knee-jerk were absent. There was at times involuntary contraction of the paralyzed muscles. No response could be obtained to the faradic current except in the quadriceps and adductor muscles on the right side and the quadriceps alone on the left. The examination for sensation was not perfectly satisfactory on account of the nervous excited condition of the patient. The loss and impairment of sensation are noted below. She had large sloughing bedsores over the sacrum and over the ilium on the right side, and over both external malleoli. These developed very rapidly after paralysis. The patient had been on antisyphilitic treatment.

On September 13, 1895, the patient began to recover power, but marked wasting in the muscles of the lower extremities was present. Muscle-jerk was normal and knee-jerk was plus. Ankle-clonus was present on both sides. She used her lower extremities freely but had not power enough to stand. Sensation was the same as when first examined.

Examination showed a number of scars about the head and neck; one of these was a linear scar on the forehead one inch long, another a depressed scar at the margin of the forehead and hair. Her lungs were normal; the second sound of the heart was accentuated; the walls of the arteries were thickened.

Tenderness was present over the muscle on each side of the spine beginning at about the tenth dorsal vertebra and extending downward.

The left pupil was larger than the right; both responded to light and accommodation.

In October it was noted that both legs were contractured. Knee-jerk and muscle-jerk were plus; front tap, ankle-clonus, and toe-jerk were present. She had isolated areas of anæsthesia in the lower extremities. She improved greatly after admission both in motor and in sensory symptoms.

The history above given chiefly represents her condition when first admitted, or at least when her paralytic and sensory troubles were at

their height, within a week after her admission. At that time the motor paralysis in the lower extremities was complete, the lower abdominal muscles being also involved. Anæsthesia to the different forms of sensation—touch, pain, and temperature—was complete up to the line where she complained of the girdle sensation. About this position there was a hyperæsthetic band, just above that of anæsthesia. The other phenomena present were as given in the history. This patient was at once placed upon active syphilitic treatment, potassium iodide and mercuric bichloride being given. She improved steadily after a few days. In a short time the motor paralysis had improved, and the upper line of anesthesia had moved downward and become somewhat irregular. On one side the loss of touch, pain, and temperature sense reached to about the middle of the side, and above that sensation was blunted to temperature only, to a distance several inches higher. This woman had suffered pain in her back, which disappeared when the paralytic phenomena made their entry. This event is exactly similar to that which takes place in not a few cases of encephalic syphilis, the intense pain in the head disappearing almost immediately after the onset of a monoplegia or hemiplegia. The cutaneous, tendon, and muscle phenomena, the trophic disorders, the paralysis of the bladder and bowels, are symptoms easily referable to a lesion localized about the upper limits of the lumbar enlargement and in the lower portion of the thoracic cord. The fact that inequality of the pupils was present probably simply indicated some coincident involvement of oculomotor fibres or nuclei, a not uncommon occurrence even in spinal syphilis.

This case represents a comparatively common form of paraplegia of acute onset. Subsequent to the time when the above notes were taken the patient greatly improved, and was able finally to leave the hospital walking, although still with considerable paresis in the lower extremities.

While the following case may be said to properly belong to the class of acute syphilitic myelitis or paraplegia of acute onset, the period of its development extended over two weeks.

"The patient, R. M., is a mulatto, aged forty-one years. On two occasions, more than twenty years ago, he had chancres, but he has never had any of the usual manifestations of secondary or tertiary syphilis. He claims that he was not a hard drinker and was not addicted to sexual excesses of any kind. He has no

history of an injury. He comes from a long-lived family, and his general health was good until eighteen months ago, when without any known exciting cause he was taken with a feeling of soreness and weakness of the left foot, which soon extended as high as the left knee. In two weeks the right leg below the knee was similarly affected, and within a month he began to experience pain in the lower part of the back. For a few weeks before the paretic symptoms set in the left foot would now and then feel numb and cold. Gradually the loss of power in his lower extremities and the unpleasant sensations in both legs and back grew worse. Occasionally he had involuntary twitching of the legs. Four months after the first positive paretic symptoms, he began to have the sensation of a band or girdle around the waist. In six months from the first appearance of the sensorimotor symptoms in his left foot he was compelled to take to his bed. His legs were now quite helpless; he had constant dull pain in his back; the girdle sensation and a feeling of dragging in the abdomen were constantly present and very annoying; passages from the bladder and bowels were involuntary, and he had marked symptoms of abnormal reflex activity, such as a curious jerking of the knees, and retraction of the feet on slight irritation. He had no mental manifestations; no facial or ocular troubles; no symptoms whatever referred to the upper half of his body. No bedsores made their appearance. He remained in bed several months and took large quantities of potassium iodide, from which he seemed to derive considerable benefit. Severe cauterization of his back was resorted to twice, and apparently did good. Under its influence the girdle sensation disappeared, the pains in his back improved, he partially regained control of his bladder and bowels, reflex excitement largely subsided, and his legs recovered some of their strength."[1]

Hæmatomyelitis sometimes simulates subacute anterior poliomyelitis. A striking peculiarity of hæmatomyelitis is the sudden paralysis which may develop even more rapidly than in true Landry's paralysis—even in a few minutes. It usually first appears in the legs, then the arms are affected, with pain in the back and other sensory symptoms. The differences between it and Landry's paralysis are in the very sudden onset and prominent symptoms of irritation. The following are the notes of a case of this kind, with post-mortem, observed in this hospital:

"J. R., colored, aged sixty years, was brought to the hospital with the statement that he had suddenly a stroke of paralysis eight days previous, since which time he had been getting worse. When admitted he was sinking rapidly, the breathing was short and labored, the heart's action was feeble and irregular, the pulse 104, feeble and occasionally intermitting. He could not protrude the tongue beyond the lips, and swallowed with difficulty, the act being accompanied by a choking sensation and the ejection of frothy mucus. He spoke with great difficulty and complained of sharp pain in the back, between the shoulders and up and down the spine. He had no headache. The bowels had not been opened, but the urine was passed involuntarily. He had a moderate amount of power in his arms. The legs were stiff and motionless. The right axillary temperature was 102°; the

[1] Mills, C. K.: Med. Record, vol. xvi, Oct. 18, 1879.

left, 102.5° F. Every few minutes there was a spasmodic jerking of the legs and body, but no trismus. An autopsy showed minute hemorrhages throughout the gray matter of a softened cord."[1]

At the meeting of the American Neurological Association in 1892 I reported a case of fatal acute myelitis, mainly of the dorsal cord.[2] Although I was at that time inclined to regard the case of pyæmic origin, further consideration has led me to the view that it was probably syphilitic.

"This patient, with an uncertain specific history, six months before coming under observation had had a large carbuncle between the shoulders, and for months had shown some tendency to drag his feet. Four days before he was first seen he was taken with severe pains across the loins; in thirty-six hours he could not stand, and twenty-four hours later he was completely paralyzed in both lower extremities and totally anæsthetic as high as the nipples, with incontinence of urine and fæces and abolition of knee-jerks, muscle-jerks, and skin reflexes; temperature rose rapidly to 104° and 105°, with corresponding increase of pulse and respiration. For a few days his serious condition did not change much, then rapid increase of symptoms took place, and he died evidently from cardiac and respiratory paralysis. The entire course of the acute attack was about ten days, and included a first period of rapid development lasting about four days, a second period of little change or advance lasting four days, and a final rapidly fatal period of about two days. Autopsy showed acute transverse myelitis in the midthoracic region, where a shell of solid cord tissue surrounded a creamy mass. Toward both the cervical and the lumbar region the evidences of myelitis were less and less marked. Microscopical investigation showed the nervous tissue almost entirely destroyed, with distended blood-vessels, and many scattered hemorrhages."

The next case is a fair illustration of a mild, and, under favorable circumstances, nearly curable type of acute syphilitic myelitis.

"H. S., aged twenty-eight years; a seamstress; has had six children, only two of whom are living, and it is suggestive that her health was good until the birth of her first child, but since has been bad, and that with each child she has lost her hair. Seven months ago she began to have trouble with her feet and legs, principally the left leg; first a feeling of heaviness, which became quickly worse, until both her feet and legs were numb and as if asleep. For two days she had complete loss of motion in both lower extremities; in her own words, she could not even move a toe. She had also for a brief time complete loss of sensation in the legs, and trouble in passing her water, requiring to strain very much, although it did not become necessary to use the catheter. This, so far as I can obtain it, is the history of the acute onset; as I have no record of examination made at the time, I must depend upon her own statements. After a few days she began to improve and was soon able to walk, but she has never since been quite strong in her legs. Examination showed no pain or tenderness or impairment of sensation. She

[1] Mills, C. K.: Med News, vol. xlvii, Sept., 1885.
[2] Journ. Nerv. and Ment. Dis., vol. xvii, N. S., p. 657, Aug., 1892.

swayed on standing and more on attempting to walk with her eyes closed. All movements were preserved in both lower extremities, but she showed some general weakness, and especially some loss of power on the left, most marked for dorsal flexion and abduction of the foot. Knee-jerk and muscle-jerk were very pronounced, but more marked on the left; front tap present on the left, but not on the right; ankle-clonus decided on both sides; toe-jerk absent; electrical responses normal."[1]

In a previous volume of the PHILADELPHIA HOSPITAL REPORTS (Vol. II, 1893) Horwitz, under the title of "Spinal Anæmia Due to Syphilis," reports some cases of great interest, the first with an autopsy and microscopical examination. While the paper and the cases included in it form a valuable practical contribution to the subject of syphilis of the nervous system, it seems to me questionable whether the title used for his cases is accurate.

The first case, and the one most important because of the autopsy, was a man, forty years of age, who, when twenty-two years old, had a severe outbreak of syphilis. About two years before he was seen by Dr. Horwitz the first signs of spinal disease were manifested in pains in the back, legs, and arms, of a dull, aching character, which were worse at night. In the end he was completely paralyzed in both lower extremities. The thoracic and part of the cervical cord were flattened and contained areas of softening; a microscopical examination of the pial vessels showed thickening, degeneration, and in many places obliteration. In brief, in accordance with the methods followed in this paper and in those usually followed in recent works on syphilis of the nervous system, the case would be regarded as one of chronic disease of the vessels with paraplegia of acute onset, due to vascular disturbances, obliterative thrombosis, and acute softening.

Other cases observed at the Philadelphia Hospital were evidently victims of anæmia and syphilæmia, but this was probably associated with disease of vessels of all parts of the cerebrospinal axis, and in some cases probably with inflammatory disease, not only of vessels, but also of the membranes. The term cerebrospinal anæmia might be properly given to cases in which, owing to the syphilitic poison, the vessels supplying the nervous system, and especially the smaller arteries, have become diseased (but not to the point of obliteration), and in consequence the parts to which these vessels are distributed are irregularly and imperfectly supplied with blood.

The form of chronic spinal disease which has come to be known commonly as Erb's spinal paralysis is comparatively common at the Philadelphia Hospital, and during the last ten or fifteen years not a few such cases have been recorded by members of its neurological staff. These cases do not always conform closely in onset, history of

[1] Mills, C. K.: Internat. Clinics, vol. ii, 4th ser., 1894.

progress, and symptomatology to a well-defined type, but they are sufficiently similar to enable them to be recognized and classed apart, both from acute syphilitic spinal affections and the degenerative diseases. These cases, while showing great irregularities in symptomatology, have certain main features, which are slowly-developed spastic paraplegia, exaggerated tendon and muscle phenomena, disorders of bladder and bowels, and varying conditions as to sensation. Usually the condition comes on slowly, paræsthesia being prominent, and pain in special distributions not infrequent. Anæsthesias may be absent, or may be slight and found only by searching. The spastic paretic or spastic paralytic condition is the dominating feature. Not uncommonly one leg is attacked before or at first to a greater extent than the other, but after the disease has made considerable progress the difference between the two extremities in this respect is not marked. Sexual disturbance or impairment is comparatively common.

By some it is supposed that Erb presented what he believed to be a fixed or at least somewhat constant type of spinal disease, entirely distinct from the types recognized before his first publication on this subject. This is perhaps not stating the matter exactly as it should be stated. He found on examination that numerous cases, usually set down as forms of lateral or combined sclerosis, or possibly irregular posterior sclerosis, presented a somewhat uniform complexus of symptoms. A study of a number of these cases led him to the conviction that they could be separated by their history and classed according to the phenomena which they presented. These cases are invariably of syphilitic origin. In the wards of this hospital a number of cases, confirming more or less closely the descriptions of Erb, have been studied by the different members of the neurological staff.

In an article on "Syphilis of the Nervous System," in a *Text-Book on Nervous Diseases, by American Authors*[1] (Phila., 1895), Dercum speaks as follows of the type of syphilitic spinal paralysis under consideration :

[1] As one object of this paper is to call attention to the work on syphilis of the nervous system contributed by those connected with the Philadelphia Hospital, reference might be made to this excellent article by my colleague, Dr. Dercum. The different forms of syphilis of the nervous system are considered in a condensed and practical manner, and several excellent illustrations of types of intracranial syphilis from patients studied in the wards of the hospital are given.

"According to Kuh, the disease has its origin, in all probability, in syphilis of the vessels of the cord in the dorsal regions, the lesions in the cord being secondary and involving mainly the lateral tracts, and slightly invading the posterior columns. The recognition of this type is exceedingly important, as, according to Erb, marked improvement is apt to occur. Kowalewsky, who has also studied Erb's symptom group (for it cannot be termed a type), concludes that it is quite common, that it belongs to the ages of thirty to forty-five years, and that it is most common in the male sex.

"As is well known, ataxia is now and then present in syphilis of the cord. At times this ataxia is associated with exaggerated reflexes, so that a condition is presented similar to that which is met with in ataxic paraplegia or combined sclerosis. In other cases, again, the ataxia may be associated with absence of the tendon reactions, and the clinical picture may still further resemble that seen in true locomotor ataxia in the presence of an Argyll-Robertson pupil.

"When we recall the fact that the virus of syphilis expends itself mainly upon the membranes and blood-vessels, it is not surprising that most varied clinical pictures should present themselves. Not a single symptom can be taken as constant for all cases. Even the spastic character of the gait may be lacking. Instead of flaccidity, contractures may be present, and under certain conditions the knee-jerks may be absent. Indeed, Oppenheim goes so far as to maintain that Erb's type is only a stage of meningomyelitis."

E. H., aged thirty-five years; white; single; seaman; was admitted to the hospital June 7, 1893.

On June 17, 1892, he was taken with a weakness in the left leg, and with inability to pass urine. The weakness increased and he was not able to walk. He probably had overflow of retention at that time. Three months later he had sensations of numbness and coldness in the right leg. He had had no trouble above the waist before or since that time.

He had chancre three years before his first symptoms, and in 1894 he had alopecia; he had three attacks of gonorrhœa in nine years. He was a moderate drinker. He was never injured.

For nearly one year he grew steadily worse. He had paralysis in the left leg, and numbness in the right leg increased. His urine dribbled away every half hour. Jerking of the legs was a prominent symptom. Notes were made in 1898 as follows:

His mental condition is good. Tactile and pain senses are preserved on the legs as high as the knees. The temperature sense is affected on the right leg between the knee and the ankle. Hot and cold test-tubes are appreciated only as touches. The senses of location and of pressure are good. There is no retardation

of sensory conduction of touch and pain. The cutaneous reflexes are much exaggerated. In the left leg quadriceps-jerk and knee-jerk are plus. Front tap, ankle-clonus and toe-jerk can be elicited. The deep reflexes on the right are the same as on the left, except that they are much less marked, and paradoxical reaction is present.

Station is somewhat affected; he sways a little on standing; his gait is paretic spastic. The loss of power is greater in the left leg than in the right.

W. F., aged fifty years; white; single; teamster; was admitted to the hospital May 24. 1893.

In October, 1892, he found that he could not urinate without difficulty. He had pain in the bladder and had to be catheterized. Two days later he had twitching in the toes of both feet, and twitching, numbness, and tingling in both feet. He continued to walk and to work for six weeks, but he had a peculiar, unsteady, jerky gait, which he described as "like a horse with a string halt."

In December, 1892, he went to the surgical wards and remained six weeks to be treated for bladder and kidney trouble. He was discharged and readmitted to the nervous wards May 24, 1893. In the meantime his legs had become paræsthetic and somewhat contractured. He has remained in the hospital ever since. In 1898 his condition was as follows: Tactile and temperature senses are preserved, and the conduction of all retarded. It takes several seconds for him to appreciate a prick or hot test-tube applied to his feet. The plantar cutaneous reflex is much increased; at times there is violent response from the sole of the foot before he appreciates the sensation. A slight touch at times causes violent jerking and twitching of the limbs. In the left leg quadriceps- and knee-jerks are much exaggerated. Repeated taps on the patella cause clonus. Front tap is doubtful; ankle-clonus is doubtful and not always obtained; toe-jerk was marked, the retraction of the foot on the leg, the leg on the thigh, and the thigh on the pelvis being of a positive character. Paradoxical contraction was not present. In the right leg the responses were much the same, ankle-clonus being more marked.

The hospital records of the two cases just noted are much more voluminous than the statements given, but these record the main features of the cases. These patients have been well known at the hospital for years. On one or two occasions they have gone out and returned. The first patient improved decidedly for a time and then relapsed, and since has remained in an almost stationary condition. The second patient up to 1898 had become progressively worse, but his descent was extremely slow, and for a long time the symptoms would be almost stationary. Both patients received from both the writer and his colleagues most energetic antisyphilitic treatment. The only form of specific medication not used was that of mercury by hypodermic injection.

P. McA., aged fifty-three years, a laborer, was admitted to the hospital March 28, 1894.

His complaint upon admission was weakness and lameness in legs. He had

gonorrhœa three times. No history of syphilis could be obtained. He used alcohol to excess.

His disease began by weakness and stiffness in the legs, especially marked in the knees. This progressively increased, when he became unable to walk for any distance without difficulty. His toes would catch in walking and he would stumble. He never had any pain either in his legs or body. He had no difficulty in micturition, and his bowels were regular. No sensory symptoms were demonstrated. On admission the pupils responded well to light and accommodation. The dynamometer showed a grip of 80 for the right hand, 71 for the left. His knee-jerks were plus. Ankle-clonus, jaw-jerk, and biceps-jerk were present. His gait was decidedly spastic.

Examination by Dr. J. H. Lloyd, March 17, 1897, showed atrophy of one of the pectoralis major muscles. The patient claims to have had this condition ever since he can recollect. The body of the muscle has entirely disappeared, and is in marked contrast with its fellow, which is unusually well developed.

On elevating the arm a band of apparently fibrous tissue seemed to be all that is left of the muscle. This stretches from the third rib. The pectoralis minor muscle seems also to be entirely gone. Faradism fails to excite the slightest response. A most remarkable fact is the very slight degree of loss of power in the movements of the arm. There is, however, some slight inability to forcibly flex the arm across the chest.

On December 1, 1898, he was again examined. Jaw-jerk was absent. Knee-jerks were plus. Biceps- and triceps-jerk were also absent. Slight patellar-clonus was present. Muscle-jerk and front tap were present. Ankle-clonus was marked on the left side, less so on the right. Sensation was good. The movements of the muscles of the lower part of the face were slightly limited.

ANOMALOUS AND ABERRANT FORMS OF SPINAL SYPHILIS.

Spinal syphilis, either of the acute or the chronic type, often shows itself in abortive, imperfect, or aberrant forms, the syndromic irregularities indicating sometimes a difference in the pathological nature, but often much more decidedly in the topographical distribution of the lesions. Some of the cases, especially those which have been discussed under the title of paraplegia of acute onset or acute syphilitic myelitis, are very largely myelic, the lesions being mainly confined to the vessels and substance of the spinal cord, although it would be difficult to find a single case in which the inner membranes were not in a slight degree implicated. The disease is probably always meningo-myelic, cord and membrane both being involved, although primary lesions and secondary degenerations in the cord are present in larger portion than is membranous inflammation. A gummatous lepto-meningitis without myelitis is theoretically possible, and occasionally such is present without much myelitis, but on the whole these cases are rare. These differences in relative inflammation of cord and

environment cause some of the irregularities in symptomatology, but they are more frequently caused by the peculiar topographies of the lesions. One side of the cord is affected more than the other; one side is affected to a higher level than the other; the thoracic and upper lumbar cord are most involved, or the thoracic, cervical, lumbar, or even sacral region in rare cases may bear the brunt of a syphilitic attack—and according to the site and extension of the lesion will of course be the symptomatology. These anomalous, or at least irregular, forms of spinal syphilis will be referred to after cerebrospinal syphilis is considered, for the reason that often the spinal varieties of syphilis become in some way associated with cerebral types. Cases simulating Brown-Séquard paralysis, triplegias, etc., will then be briefly discussed.

SYPHILITIC SPINAL TUMOR, MENINGEAL AND INTRAMEDULLARY.

With the exception of the cases of syphilomata of the cervical dura and cervical hypertrophic pachymeningitis already referred to, I have seen very few instances of isolated syphilitic tumors of the cord, and I have not at present command the records of such cases. Mickle[1] writes of these cases as follows:

"The gummata may be numerous and small. We are only referring to those easily visible to the naked eye and forming small tumors. Numerous microscopical cellular collections in the walls of meningeal vessels and in those of the cord are really buds of possible future gummata; but these are not at the present moment under consideration.

"More frequently there is one gumma, and of fair size, sometimes two or more of similar size. The solitary gumma is apt to affect, more or less, one side of the cord, especially the posterior column and the lateral, and thus may give rise, more or less completely, to the symptoms of Brown-Séquard's paralysis—namely, that due to a transverse lesion destroying at some level one lateral half of a transverse section of the cord. Gummatous meningitis, or a gumma even when distinctly meningeal in origin, may, by pressure, produce the same effect as if seated in the cord."

[1] Brain, vol. xviii, Lond., 1895.

SYPHILITIC DISEASE OF ISOLATED ARTERIES AND VEINS, CAUSING FOCAL SOFTENING.

From what has already been said in discussing spinal syphilis, widespread disease of the vessels of the spinal cord is common in the subject of syphilis. In rare cases such disease is limited to special arteries or veins of considerable size, or if, strictly speaking, it is not so limited, the disease is chiefly manifested in such isolated vessels. When these are arteries a thrombosis may eventually take place, and a focus of softening result. A few cases of the Brown-Séquard type, or somewhat like this type of spinal paralysis, have been reported and have been shown by autopsy to be due to focal softening. A few cases of other clinical types are also on record. Instead of focal softening, a small hemorrhage from a ruptured artery may be found, but arterial occlusion causing softening is more frequent. Syphilitic disease of the coats of the veins leads to rupture and hemorrhage, a subject to which too little attention has been paid. Even embolism of the cord may occur, but it is rarer than even hemorrhage or thrombosis.

INTRACRANIAL SYPHILIS—SYPHILIS OF THE BRAIN AND ITS MEMBRANES.

"Considering the subject from the pathologic side, perhaps no better practical classification has been made than that of Bramwell, who divides encephalic syphilitic lesions into (1) intracranial nodes with or without meningitis; (2) gummata; (3) lesions of the large or medium-sized blood-vessels, chiefly obliverative endarteritis; (4) diseases of the minute vessels, in many instances periarteritis rather than endarteritis; (5) localized encephalitis in the neighborhood of gummata and meningeal lesions; (6) inflammation of the intracranial portions of the peripheral nerves; and (7) meningeal inflammations with gummatous deposits (?). Almost every one of these classes of lesions will be illustrated by our cases."[1]

Oppenheim's classification is both topographical and pathological. The subdivisions are (1) basal syphilitic affections; (2) syphilitic basilar meningitis with arterial symptoms; (3) syphilitic affections of the brain convexity; (4) meningitis and syphilitic circumscribed meningo-encephalitis of the convexity; (5) diffuse meningitis and meningo-

[1] Mills: Med. News, vol. lxvii, Nov. and Dec., 1895.

encephalitis of the convexity; (6) gummata; (7) primary syphilitic neuritis of the cranial nerves and multiple syphilitic root-neuritis; (8) primary syphilitic arteritis; (9) syphilitic encephalitis; (10) cerebro-spinal syphilis; and (11) cerebral affections called forth by hereditary syphilis.

The classification of intracranial syphilis which will be followed in this paper is slightly modified from those of Oppenheim and Bramwell. The subject will be considered under the following heads:

(1) Syphilitic osseous and periosteal disease; (2) intracranial gummata; (3) basal gummatous meningitis and meningo-encephalitis; (4) lesions of vessels and their results; (5) meningitis and meningo-encephalitis of the brain convexity; (6) neuritis of the cranial nerves.

SYPHILITIC OSSEOUS AND PERIOSTEAL DISEASE.

Swellings or nodes due to syphilitic disease of the periosteum, or both the periosteum and the bone, may give rise to symptoms that simulate closely those of meningitis or tumor, and, indeed, meningitis, tumor, or softening due to vessel-disease may be associated with such nodes. These are often extracranial, and it is by no means uncommon to find intracranial nodes present in the same case with the extra-cranial swellings. The prominent symptoms will usually be headache with evidences of pressure and irritation, according to the site of the lesion. When such nodes are prefrontal and large, somnolence and mental hebetude may be marked. Paresis or affections of the special senses may be present. Occasionally these periosteal swellings are situated along the course of the cranial nerves or around their foramens of exit, and thus may give rise to cranial nerve palsies, anæsthesias, and neuralgias. As syphilitic nodes, either intracranial or extracranial, when efficiently treated, have a more favorable prognosis than some of the varieties of encephalic syphilis, it is important to bear in mind the probability of their existence. They sometimes melt away under active treatment with mercury and iodides.

In the Pathologic Museum of the Philadelphia Hospital are two specimens of osseous and periosteal disease. One of these is from a patient who was for many months under my care. Syphilitic periostitis, ostitis, and necrosis finally caused the large irregular opening seen in this calvarium. The nervous system of this patient was also attacked in various other ways by syphilis. The other is a specimen

of syphilitic ossifying periostitis. While some cases of periosteal and osseous syphilis are amenable to early and active treatment, this specimen shows the permanent ravages that may be produced by such diseases.

INTRACRANIAL GUMMATA.

While syphiltic lesions of the dura are usually diffuse and take the form of a more or less extensive pachymeningitis, occasionally a recent or old gumma is found to have originated from the dura. More commonly this membrane is invaded by an endostosis, or dura and pia are agglutinated together by inflammation, and the growth which seems to arise from the pathologically united membranes in reality has sprung from the vessels and the connective tissue of the pia. I have seen but few instances of syphilitic tumors of the dura, but the following was recorded by me several years ago. In this case the very circumscribed growth was probably a gumma which has undergone complete fibrous change. The influence of traumatism in fixing the site of this growth and, perhaps, in determining the existence of an isolated gumma is not improbable.

J. J., aged thirty-two years; white; born in Scotland; laborer; was admitted to the hospital August 9, 1888, complaining of severe headache and sleeplessness, which had existed for three months. He was obstinately constipated and had lost appetite and flesh, although he was still a large, muscular man. His breath was offensive; his urine was normal. Knee-jerk was normal. No paralytic symptoms were present. The right pupil was rather larger and responded to light more slowly than the left. Eight years before, while serving as a policeman in Scotland, he had been struck on the back of the head with a blackjack, was rendered unconscious, and remained so for a considerable time, and after this he suffered considerably with headache, but finally recovered. He attributed his attack to this accident and the abuse of alcohol. He denied syphilis, but admitted having gonorrhœa fourteen years before admission. At the time of his last attack he was working for a plumber in different parts of the city, until his headache became so severe that he had to quit. He located the pain over the right eye. It varied in intensity, always being worse at night. On admission, his temperature was 100°; pulse, 50, and respiration, 18. The next morning his temperature was 97°; pulse, 48; respiration, 18. He was extremely restless, tossing about, but perfectly conscious. His urine was scanty and high-colored, and passed only at long intervals. He was quieter and somewhat dull the third morning, and the dullness increased, so that by 10 A.M. he had to be aroused to answer questions, and then answered slowly but intelligently. Two hours later he was completely comatose, with labored respiration. At 1.30 P.M. a decided change was noticed; his breathing became stertorous and slow; his pulse, which had been above 48, increased to 130; respiration dropped to 10, then 6, and, after two or three ineffectual efforts at inspiration, breathing stopped. The pulse continued to beat for fully five minutes

after the cessation of respiration. Artificial respiration was tried, with no effect. The autopsy showed thickening of the skull about the size of a silver dollar, beginning one inch above the line of the external meatus; the skull at this point was strongly adherent to a tumor or thickening of the dura. The mass was flat, almost circular, one eighth of an inch in thickness, tough and whitish-yellow in appearance. The dura and pia were agglutinated and adherent to the brain beneath the growth, and both skull and brain were bruised and eroded. The growth was so situated as to cross the fissure of Sylvius over portions of the posterior central, inferior parietal, and first temporal convolutions, its anterior limit being in the middle of the latter. The lesion was strictly meningeal, but in enlarging had slightly invaded both the skull and the brain substance. Subsequent examination showed the growth to be syphilitic."[1]

W. B. McC., aged fifty-six years; white; laborer; family history negative; had gonorrhœa thirty years ago. Thirty-two years ago he had a scalp-wound on the left side of his head.

In October, 1894, he had a severe fit, with twitching of the left arm and a feeling like pins and needles in the hand, this being followed by unconsciousness and general convulsion. The convulsion was followed by numbness for two to three days.

One of the records is as follows: Station, good; sensation and motion, normal; pupils, normal; knee-jerks, minus.

A note was made on August 7, 1895, that he had a severe fit. Spasm was most marked in the left arm, leg, and face; he was not entirely unconscious, but was somewhat dazed. Tactile sensation was blunted in the left arm and in the left leg. Five minutes after the attack the arm and leg were nearly paralyzed, and the face drooped on the left side. Ten to fifteen minutes later the limbs had regained their power. After the attack his arm and leg felt numb. On October 4, 1895, the left side of the patient's head was struck accidentally, which caused one of the left-sided convulsions. The patient said that after the blow a flash went down his left side. He had headache almost continuously, and hearing in the left ear was affected.

Some weeks after the above record was made this patient died, and autopsy showed a tumor, probably of syphilitic origin, growing from the dura and compressing, but not destroying, the upper portion of the motor area on the right side.

A formed and more or less firm gumma growing from membranes or vessels, and invading any portion of the cerebral cortex or both cortex and subcortex, gives general symptoms which differ only in extent from those of a localized gummatous meningitis. The syphilitic tumor very commonly, almost invariably in my experience, causes pain of a severe and sometimes agonizing character, the pain usually having marked nocturnal and other periods of exacerbation. Pain increases as the growth extends up to a certain time, when it may almost suddenly decrease or even disappear. This disappearance is usually accompanied by the occurrence of a monoplegic or hemiplegic attack, or by one indicating a new destructive focus in some

[1] Mills, C. K.: Univ. Med. Mag., Nov., 1889.

region, lesion of which furnishes marked local symptoms. As cerebral convexity surface lesions are usually of the motor region, in most cases the attack is one of paralysis. In addition to pain the most common general symptoms of gummata are the other well-known phenomena of brain tumor of whatever nature or location, namely, vertigo, recurring attacks of nausea and vomiting, often associated with exacerbations of headache and vertigo, optic neuritis, and mental irritation or disturbance. Vertigo of some description is exceedingly common in cases of gummata of the convexity, doubtless for the reason that such growths are usually meningeal in origin. Irritation of the trigeminal nerve-branches in the dura reflected to bulbar centres is the most reasonable explanation of this vertigo. The same explanation holds for the nausea and vomiting. When these symptoms are absent or are not prominent the dural nerves have probably escaped extensive implication. While optic neuritis is a common symptom of gummata of the meninges and cerebral surface, it is absent in a fair percentage of cases, and its absence should not therefore decide against the diagnosis of this as of other forms of tumor. I have noted its absence in a number of cases of limited dimensions in the motor and post-motor regions of the convexity.

It is scarcely necessary to enumerate the focal symptoms of syphilitic lesions of the convexity, whether due to formed gummata, unorganized meningitis, or to areas of softening from occlusion. These symptoms are those which are explicable on the known functions of the different cerebral centres and zones. Focal lesions of the prefrontal region give psychical symptoms of a special character: if far back on either side they may afford oculomotor symptoms with a tendency to conjugate deviation; if on the left side, motor agraphia may be present if the posterior portion of the second frontal convolution is involved, or motor-aphasia if the disease has attacked the third frontal convolution; and finally, as the result of pressure, increasing paresis may be present. In rare cases in which the orbital surface of the frontal lobe is attacked, olfactory symptoms, hemianopsia, or even third or sixth nerve symptoms are occasionally present as the result of extension of the lesion. The symptoms of focal lesion of the motor zone are so well known and have been so frequently repeated that it will only be necessary to say here that in so far as they are irritative phenomena they are exhibited in various forms of monospasm, or

hemispasm with particular phenomena, according to their exact location; and in so far as they are the signs of destruction or pressure they are types of paresis or paralysis, from the most local form of monoplegia involving a finger or toe or a muscular group in the face, to a complete hemiplegia. The visual, auditory, and other areas of the cerebral surface give the symptoms which are known to belong to lesions of these regions, as letter- and word-blindness, hemianopsias, and word-deafness.

The following are instances of gummata variously located and chiefly studied at the Philadelphia Hospital:

W. V., aged sixteen years, male, was admitted to the hospital about two weeks before his death. He had had occasional convulsions for over a year, and thought that he had received a blow on the head before they came on. He had a scar about an inch long in the scalp on the right side of the head, but it seemed to be superficial; no depression or cleavage of bone could be made out. On or about December 27, 1886, in trying to rise from his chair, he fell. He had noises in his head, or as he expressed it, "his head moaned." He also had vomiting at this time, and obstinate constipation; and sound and light were intensified by the pain in his head. He had some fever, and his pulse was sometimes as low as fifty a minute. The next day there was some loss of power, and he walked with a staggering gate; he had headache and cold sweats. On the third day he had diplopia, vertigo and palpitation; and he now cried out with severe pain in his head, and could retain nothing on his stomach. He also had seizures, the nature of which could not be determined. These became somewhat better but were followed by sensations of pins and needles in the arms, accompanied with great stiffness. His bowels continued to be much constipated. In the second week of his illness he lost power to some extent in his left arm, and had some depression of sensation in the right. Although during this week he seemed to be stronger, on attempting to walk he staggered and fell. He had intolerance of light at this time, especially marked in the right eye. During his fits, he was drawn to the left side, and always cried out before they came on. He had sensations of choking during the fits, and said that he was never unconscious in them. He was ravenously hungry most of the time during his illness after the first week, and bolted his food at all times. His temperature on admission was 99°, and afterward ranged between 98° and 99°. He had attacks of vomiting at irregular periods, and his bowels were much constipated, but readily moved by enemata. His eyes were somewhat immobile, making his gaze seemed fixed. His pupils were equal. He had a slight but distinct ptosis of the right eye, sometimes more marked than at other times. He could not close the left eye, nor wrinkle the left forehead. He had distinct loss of power in the left arm and hand, and also in the left leg, but not so marked. He died apparently from general exhaustion.

The dura was not adherent to the skull, but on attempting to remove it the right side was found to be strongly adherent to the pia, and the brain underneath it over the antero-frontal region, and the corresponding portion of the median surface of the right hemisphere. In removing the brain a little serous or grumous fluid exuded from this region, but no true pus. On carefully removing the dura,

a reddish-gray grumous mass was found adherent to the agglutinated dura and pia, and involving the gray matter of the brain and a portion of the white matter immediately adjacent. This was soft, and in its centre a hole or cyst appeared, or the space had been occupied by tissue now broken down.[1]

"Female, aged thirty-eight years. History of syphilis. Blows on the head. Headache with agonizing paroxysms. Top and right side of the head sensitive to percussion, and headache severest in these regions. Vomiting; vertigo. Great mental irritability. Severe left-sided spasm, beginning with twitchings in left toes and foot. Partial paralysis of right leg and arm, most marked in leg. Hyperæsthesia. Impaired sight. Choked discs. Head-temperature, taken once: right parietal region, 97.2° F.; left parietal region, 96° F.

"Gumma. Attached to the fused membranes of the right convexity. Involved the upper fourth of the ascending frontal and a smaller segment of the ascending parietal convolution, crossing Rolandic extremity. A good example of strictly cortical lesion."[2]

"Male, aged nineteen years. First symptom, headache; then vertigo. Sudden right brachial monoplegia; possibly some paresis of leg. Recovered use of arm; went to work; was kicked by a mule, and became worse. Headache and right-sided paresis returned. Increasing stupor; paralysis of right arm complete; of left leg almost; right facial paresis; ptosis of right side. Partial anæsthesia on right side of face; pain on right side. Slight clonic spasms of right arm. Paralysis of bowels and bladder in last week. Tendency to Cheyne-Stokes respiration. No vomiting. Eyes not examined.

"Gumma. A large tumor in the ascending frontal convolution, at junction of middle and upper thirds; one-third of mass on convexity of convolution, the remainder in fissure of Rolando. Smaller tumor at inferior angle of right lobe of cerebellum. Some basal meningitis with effusion."[3]

"Male, aged thirty-three years. History of syphilis. Blows on the head. Headache continuous, usually dull, occasionally severe. Occasional vomiting. Stupidity, want of energy, drowsiness. One general convulsion a few hours before death. Partial anæsthesia on the right side. Hyperæsthesia of left side. Sight impaired. No ophthalmoscopic examination. Hearing defective in right ear. Constipation.

"Gumma. One and a quarter inches in diameter attached to adherent membranes, and involving the middle portions of the first and second temporal convolutions of left hemisphere. A layer of brain substance both anterior and posterior to the tumor was softened. The tumor grazed the ascending parietal and inferior parietal convolutions. A large foyer of greenish-yellow pus was found to the inner side of the tumor."[4]

In the following case the history and the examination of the patient all point to syphilitic disease at the base, probably a gumma or gummatous meningitis chiefly in the region of the left crus. The fact that

[1] Mills, C. K.: Journ. Nerv. and Ment. Dis., vol. xiv., Nov. and Dec., 1887.

[2] Mills, C. K.: Arch. Med., vol. viii, No. 1, Aug., 1882.

[3] Mills, C. K.: Med. and Surg. Rep., vol. li, Aug. 2, 1884.

[4] Mills, C. K.: Arch. Med., vol. viii, No. 1, Aug., 1882; also (with James Hendrie Lloyd, M.D.) in "A System of Practical Medicine, by American Authors" (edited by Wm. Pepper, M.D.), vol. v, Phila., 1886.

only one of the third nerve branches, that to the levator palpebræ, is involved does not negative this localization. It is not uncommon in a case of gummatous meningitis or soft gumma to have one or more cranial nerves entirely involved, and some of the separate divisions of these nerves escape destruction or compression sufficient to give definite symptoms. I have seen almost the entire central base involved in a gummatous meningitis, and yet the cranial nerve symptoms had been limited and irregular in character. The function of a nerve can be largely maintained even in the presence of a considerable lesion of this character. Another explanation of the partial ptosis might of course be the isolated implication of the levator nucleus on one side. Although the notes of Dr. T. H. Weisenberg given below indicate a staggering or ataxic gait, my examination of this patient seemed to show that it was due to a paretic or paralytic state involving the right side more than the left. The possibility of a cerebellar lesion was considered because of the pseudo-ataxic station and gait of the patient.

A. C., aged twenty-one years; white; housewife; was admitted to the hospital October 4, 1900.

She was a patient in the venereal wards of the hospital a year ago. On admission to the nervous wards she was stupid and could state no symptoms. She was a young woman, pale and anæmic, with a vacant expression. Her pulse was slow and fair. She had an old rotary lateral curvature, the left shoulder is higher than the right, and she is chicken-breasted. The apex-beat of the heart is normal; there is a prolongation of the first sound, the murmur is transmitted to the axilla. The second sound is accentuated. Examination of the lung proved negative. The abdomen and the extremities were normal.

Her pupils dilated to the full extent with homatropin; previous to dilatation they were small and reacted to light. In the right eye the disc was slightly prominent, about four dioptres. There was a well-marked beginning neuritis above at the upper border. The left eye presented a similar condition, neuritis being not so marked. Swelling was about four dioptres. There was apparently no failure in rotation of the eyes, the external ocular muscles not being affected. There was slight ptosis of the left eye, about half of the corneal surface being covered by the drooping lid. The patient's mental condition was such that it was impossible to take fields, but certainly no hemianopsia was present.

The right side of the face was smoother than the left, and drooped somewhat. The left side of the face was slightly drawn, and in talking, crying, etc., she made use of the left side of the face more than the right. Grip in the right hand was 0, in the left, 4. She dragged the right foot, and on attempting to walk alone she staggered and fell like a person who is intoxicated. Her station was very poor. There was apparently no loss of sensation on either side. At first she appeared to be more sensitive on the right side, but she became confused a minute later and could not answer questions. The thermal sense was normal. No astereognosis was detected. Von Bechterew's reflex was present on both sides, slightly less

marked on the left. Triceps-, biceps- and flexor-jerks were plus on both sides, but a little weaker on the left. The extensor reflex and ankle-clonus were absent on both sides. The quadriceps- and knee-jerks also were present on both sides. Babinski reflex was present on the right but absent on the left, the whole foot turning up on the left, not the toes merely.

"Male, aged thirty-two years. Thrown from a horse and kicked on the head. History of syphilis; headache, severe at first, always came on at night, became less severe later. Vertigo. Defective memory, apathy. Right hemiparesis; helplessness of all the limbs before death; partial ptosis of the right side. Sensation diminished in the left side of the face and in the right limbs. Pupils small before death. Descending optic neuritis. Conjugate deviations of the eyes and rotation of the head to the right. Persistent epistaxis; tendency to hemorrhage from mucous membranes. Constipation.

"Gumma and fibroma. (1) Gumma, half an inch in diameter, distinctly limited to the left upper quarter of the pons. (2) Fibroma, no larger than a pea, between the dura and pia mater, causing a slight depression in the first temporal convolution about the junction of its middle and posterior thirds, and half way between the parallel fissure and the horizontal branch of the Sylvian fissure. Microscopical examination of the optic nerves showed a descending neuritis of subacute character."[1]

"Male, aged thirty-five years. History of syphilis. Wounded in head by glancing bullet. Headache of great severity at intervals. Vomiting at time of headache. Vertigo. Failure of memory and depressed spirits. Epileptiform attacks. In the spasms lifted up the right side of his body and worked over toward the left. Occasionally cramps in the stomach and legs. Temporal and orbital neuralgias. Descending neuritis and eventual atrophy of both optic nerves. Constipation. Died at the close of a series of convulsions, death being preceded by general paralysis.

"Gumma. A rounded mass, one-quarter of an inch in diameter, attached to the pia, just to the left of the centre of the anterior surface of the pons. The entire anterior central portion of the pons was softer and more doughy than usual. At the junction of the pons with the medulla was a recent hemorrhage, which had spread downward to about the middle of the latter."[2]

BASAL GUMMATOUS MENINGITIS.

My experience would lead me to coincide with Oppenheim's statement that of all forms of cerebral syphilis the diffuse basal gummatous meningitis is the most common, but I would qualify this assertion by saying that while this is true it is uncommon not to find this form of meningitis associated with other and usually somewhat numerous or extensive, syphilitic lesions in other parts of the brain. In the cases to which I have reference the gummatous meningitis of the base is the chief lesion, and cranial nerve and other basal symptoms

[1] Mills, C. K.: Journ. Nerv. and Ment. Dis., July, 1881.
[2] Mills, C. K.: Brain, Jan., 1880.

dominate the clinical picture; but evidences of convexity and subcortical disease may often be found by searching, and if not discovered during life are not infrequently seen to be present on the post-mortem table. I have put on record a number of these cases, some with post-mortem examinations, and my colleagues in the nervous wards of the Philadelphia Hospital have contributed others to the literature of neurology.

In one such case recorded by me,[1] a woman, when admitted to the hospital was so demented that papers were made out for her admission to the insane department. She had at least one convulsive seizure. Her symptoms on the first recorded examination were oculomotor paralysis with slight paresis of the right side of the face, some hesitation of speech, mild dementia, and a tendency to undue emotionality. In another case recorded in the same paper and referred to in my treatise on diseases of the brain and cranial nerves the patient exhibited right oculomotor paralysis with paresis of the left leg, mental symptoms, and a long apoplectiform period preceding death. The autopsy showed fibroid meningitis, diffuse endarteritis, multiple thrombosis, and localized softenings.

J. F., aged fifty years, a laborer, was admitted to the hospital November 5, 1898. Three years before he had chancre, followed by secondary eruption and sore throat. His present illness began in August, 1898, when he was attacked with a severe headache, was unable to work, and after a month spent in bed one day had a chilly sensation in his head, passing down the right side, with loss of power, which steadily increased. Pain in his head and back ceased when paralysis began. Three weeks ago a papular eruption appeared, principally on the left side of his face, localized more especially in the neck and right ear. The eruption was not painful, and was covered with whitish scales, small, not itching. An eruption exists on his legs which is non-inflammatory, dull red in color, and symmetrical.

There is marked limitation of movement of the right leg, loss of power of flexion and movement as compared to the left, and the same condition in the right arm, with slight contracture and spasticity. Grip is markedly less in the right arm. There is slight involvement of the muscles of the right side of the face.

Patellar- and ankle-clonus are present on the right side. Knee-jerk is increased on the right side. Front tap is present. The plantar reflex is plus. Other superficial reflexes are present. Biceps- and triceps-jerks are present on both sides, more marked on the right. No jaw-jerk is present. Tap on the right arm causes flexion of the fingers, more marked than on the left. The patient is able to twist his mouth to the left, but not to the right. He is not able to close his eyes fully. Sensation to touch appears normal over the entire body, but the patient does not easily distinguish sharp from dull points on the paralyzed side.

[1] Medical News, vol. lxvii, Dec., 1895.

Ophthalmoscopic examination by Dr. de Schweinitz showed pupil reaction to be normal. In the right eye the disc was pallid, the veins about normal, and the arteries contracted. Both arteries and veins show marked perivascularitis. The condition of the left eye is exactly the same, but the perivascularitis is more marked. The right pupil is round, the left somewhat oval.

To the physical examination add that at the base of the right lung, about the posterior axillary line, a small area of dullness exists and a few crepitant râles are heard.

Now and then highly interesting cases showing circumscribed lesions with special localizations are observed. The syphilitic disease is usually diffuse in these cases, but its chief force is exerted upon some limited region, hence the restricted nature of the clinical phenomena presented. Sometimes we see a complete, or it may be only a partial, internal or external ophthalmoplegia, or a partial internal may be associated with a partial external ophthalmoplegia, or varieties of complete and partial ophthalmoplegias may be combined, or any variety of ophthalmoplegia may be combined with any other partial or complete forms of cranial nerve paralysis. While the sixth nerve is not infrequently separately attacked, it also shows a tendency to enter into special combinations. Clinical phenomena referable to the seventh or the fifth nerves, and especially the former, may be associated with affections of the ocular nerves.

The following is an illustration of a probable case of gumma involving a large number of the cranial nerves of one side:

L. D., aged twenty-six years, a mulatto; had rheumatism about nine months ago. Seven years ago he was struck with a bar of iron on the top of the head. No other history of importance was obtained. Four months before admission he began to have frontal and occipital headache, which gradually grew worse. One morning he found the right side of his face entirely paralyzed, and he had a ringing sensation as if his right ear was "plugged up." On admission this patient was found to have complete paralysis of all the muscles of the right side of the face, including the muscles supplied by both the seventh nerve and the motor distribution of the fifth. No deviation of the tongue was present. Anæsthesia was present on the right side of the face, extending from the median line to the angle of the jaw, and from two inches above the hair line to the point of the chin. The conjunctiva of this side, also, was totally anæsthetic, as was the right half of the tongue. No part of the body other than the face was anæsthetic. Taste was entirely lost on the right side of the tongue, both at the tip and posteriorly, showing implication of both the chorda tympani and the glossopharyngeus. The electric reactions were those of degeneration in the affected muscles. A watch was heard by the right ear at about two and a half inches, and by the left at eighteen inches. A fine haze appeared to be spread over the right cornea, which, on close examination, proved to be due to numerous small points. A beginning

neurokeratitis was evidently present. Under large doses of potassium iodide the patient made considerable improvement; in particular the area of anæsthesia decreased, the neurokeratitis passed away, and there was considerable improvement of the paralyzed muscles.

In this case a large gumma probably occupied the lateral aspect of the pons and the upper portion of the post-oblongata. The abducens nerve escaped because of its more median position.[1]

W. C., aged twenty-one years; when nineteen years old had gonorrhœa, and three months before admission was treated for a painless bubo. He also had a slight eruption on his face. A few months previous to his admission his employer noticed that he showed a lack of usual energy and ambition, and that he had become careless, although he continued to work up to a day or two of admission.

He was brought to the hospital in a semi-conscious condition without evidence of injury. His right conjunctiva was injected, and the pupil of that side was much smaller than the left; both pupils reacted to light. Blood-clots were in his nostrils. His limbs were not paralyzed and there was no disturbance of sensation. He had incontinence of urine and fæces. Shortly after admission he was examined by Dr. G. E. de Schweinitz, who reported as follows: Complete paralysis of the right external rectus; the eye does not move past the median line; excursion is good in all other directions. Reaction of the pupil is normal. There is a slight opacity of both right and left cornea, probably the result of phlyctenular keratitis. In the right eye the disc is pallid, but not atrophied; the vessels are normal in size; no hemorrhages are observed. Hypermetropia 7 D in the left eye, but not as marked in the right. Epiphora (paralysis of Horner's muscle) is marked. Form field is normal. About one month later Dr. de Schweinitz again reported as follows: Vision in the right eye is one-fifth; in the left, one-fourth. Paralysis of the external rectus is as marked as on the previous examination; excursion to the outer side is nearly double that of the inner. Upper deviation is much greater. Form field is normal. The left eye has a conical macula. The disc is pale. No retinitis or optic atrophy is observed. The vessels are about normal in size.

This patient was during different terms of service under the care of Dr. J. H. Lloyd and the writer. Dr. Lloyd has recorded his case in the *Twentieth Century Practice of Medicine*, Volume XI, in the following notes:

"In some comparatively rare instances paralysis of the sixth nerve is associated with polyuria or diabetes insipidus. This is possibly due to its nucleus in the pons being contiguous to the diabetic centre in the floor of the fourth ventricle. This curious association is seen in a case at present under my care in the Philadelphia Hospital. The patient, a young man about twenty-four years of age, has had total paralysis of the sixth nerve accompanied with great polyuria. The history of his case is as follows:

"W. C., aged twenty-one years; white; male; had a history of syphilis. Before admission to the hospital he had shown mental changes of slight degree, such

[1] Mills, C. K.: Med. News, vol. lxvii, Dec., 1895.

as inattention to work, etc. He was brought to the hospital in a semi-conscious state. There was no paralysis of motion or sensation. His symptoms since his admission have been as follows: Complete paralysis of the right sixth nerve; slight dropping of the right side of the face; no involvement of the fifth or of other cranial nerves, except the sixth, and possibly the eighth, as there is some dullness of hearing; slight staggering gait, with a tendency to go to the left; marked polyuria. The examination of the eyes revealed no changes of importance in the eye-grounds. Later the patient had several apoplectiform crises, with slight clonic spasms in the right upper and lower extremities. About three months after admission the paralysis of the sixth nerve entirely disappeared, and was replaced still later by a paralysis of the third nerve. The upper eyelid drooped, the eyeball was turned outward, and the pupil was dilated. There was diplopia. The patient now presented anosmia. The polyuria, a daily record of which was kept for many weeks, was marked by a daily flow of from fifty to as high as one hundred and thirty ounces. There was no sugar nor albumin in the urine. Progressive emaciation set in until the patient had the appearance of advanced muscular atrophy."[1]

It will be noticed that Dr. Lloyd speaks of the disappearance of the sixth nerve paralysis and the appearance of third nerve paralysis; in connection with this the report of Dr. Charles A. Oliver, made between eight and nine months after Dr. de Schweinitz's last report above stated, is interesting. In the left eye good movements of the external and the internal rectus muscles. The left superior does not act promptly. The right internal rectus is paralyzed. The right eyeball deviates externally about 45, when looking at the median line with the left eye. When attempts are made to rotate the right eyeball downward it moves outward. When similar attempts are made to move the eyeball upward there is a corresponding movement outward.

The cranial nerve conditions in the following case were paralysis of the left abducens, with dilatation and immobility of the right pupil, and slight neuroretinitis.

The patient, J. D., aged thirty-six years, had a history of gonorrhœa, syphilis, and alcoholism. On admission he had a marked internal squint of the left eye. The right pupil was larger than the left, and did not respond to light. From the statements of the patient and the records furnished by Dr. Oliver the following facts were learned: Shortly before his admission to the hospital he had occasional spells of dizziness and some frontal headache. He had almost complete paralysis of the left external rectus muscle, and associated with this was slight dilatation of the right pupil, with almost total immobility of the iris to light. No disturbance of accommodation was present. The left iris was mobile to light and accommodation. Slight neuroretinitis was also present, and vision was reduced to one-seventh and one-ninth. Under fifty grains of potassium iodide, three times daily, the patient's vision became practically normal, the left internal squint nearly disap-

[1] Lloyd, J. H.: Twentieth Century Practice of Medicine, vol. xi.

peared, and the general condition of the patient greatly improved. The dilatation of the pupil remained.

This is clearly a case in which the foci of syphilitic disease attacked the root-fibres or trunk of the sixth nerve and the nucleus or root-fibres for movements of the iris. A nodose peri-arteritis or a gummatous meningitis, or both conjointly, might account for the phenomena.[1]

Occasionally one or more tumors at the base are found in the same case with a diffuse basal gummatous meningitis. One of my early cases was of this character, and is recorded in Pepper's *American System of Practical Medicine*, Volume IV, in the table of cases of brain tumor. The patient was a woman, aged thirty-five years, who had the typical general symptoms of brain tumor—agonizing headache, vertigo, nausea and vomiting, irritability, etc. She also developed a well-marked hemiplegia, paralysis of the face being complete, and a left internal strabismus. She had difficulty in enunciation. Sensibility was diminished in the left leg. Later there was hyperæsthesia and great pain in the paralyzed limbs. Conjunctivitis and necrosis of the cornea of the left eye were present, and the conjunctiva and cornea were insensitive. Sight was impaired, and descending optic neuritis was present. Hearing was impaired on the left side. Smell also was defective. Perspiration was profuse, being especially marked on the right side. Constipation was present. The post-mortem showed a twin tumor in front of the chiasm, and a diffluent and softened condition of the base backward from the position of the tumor. The case was apparently one in which a tumor had caused partial destruction of the circle of Willis, with necrosis, and in which also some gummatous meningitis was present. The symptoms were indicative of inflammation, both of the cranial nerves and of motor and sensory tracts and other regions at the base.

LESIONS OF LARGE AND MEDIUM-SIZED VESSELS AT THE BASE AND THEIR BRANCHES (INCLUDING RESULTING SOFTENINGS AND HEMORRHAGES).

Viewed from the pathological side, several classes of hemiplegia, monoplegia, and aphasia occur as the result of syphilitic disease of the basal arteries and their branches. Even the vessels of the circle of Willis, and especially the medicerebral, are likely to suffer; but in a

[1] Mills, C. K.: Med. News, 1895.

large number of cases the branches of the medicerebral and other of the larger subdivisions of the vessels of the base are the seat of the most marked disease. In some cases the weakened walls of the vessels give way and a hemorrhage occurs, but it is much more common for the lumen of the vessel to be gradually occluded by endarteritis, a clot finally closing the scarcely-open vessel, the result being necrobiosis of the tissues supplied by that vessel. In other words, syphilitic thrombosis with resulting softening is the commonest cause of the paralytic and aphasic affections connected with this form of syphilis at the base. The vessels which pass into the cerebral substance in the perforated spaces are frequent sufferers. A large percentage of the hemiplegics in the Philadelphia Hospital has had this variety of syphilitic arterial disease.

The diagnosis of intracranial syphilis is helped out in these cases by a study, not only of the history of the cases, but also of the manner in which the affections occur, and above all by the succession, multiplication, or recurrence of clinical features.

J. B., aged thirty-seven years, a butcher, was admitted to the hospital November 10, 1898.

The attack began November 9th, when the patient walked into the house unable to speak, and laid down upon the floor, where he remained until the following afternoon. At that time he was picked up, placed in a chair, and as the expression of his eyes aroused suspicion, a physician was called, the fact of right-sided hemiplegia discovered, and the man was sent to the Philadelphia Hospital, where he arrived at 8.30 P.M. From the man's brother was obtained the history of an attack of paralysis of the left leg two years previous, the paralysis improving after a little so that the patient was able to walk with a cane. He also had shooting pains, which he referred to the affected leg. The brother gave the history of venereal disease in the patient several years before, and said that after the paralysis the patient lost ambition and energy.

The following notes of his condition on admission were made: A well-nourished adult male, not able to articulate. The eyes deviate to the right, but the eyeballs move freely in all directions. The pupils are evenly dilated and respond to light. The left angle of the mouth is slightly lower than the right, and there is difficulty in opening the mouth. The tongue is not protruded. The pulse is slow, full, and steady. The first heart-sound is loud and strong, the second aortic accentuated. No murmurs are detected. The lungs are normal. Paralysis of the right side is complete, involving the face. The right eyelids in working do not close perfectly. On the inner side of the left leg is a punched-out scar, as of a recent ulcer. Knee-jerks are absent on both sides. Plantar reflexes on both sides are plus. No clonus is present. Sensation cannot be tested thoroughly because of the patient's inability to articulate and his failure to respond readily. The patient was catheterized, and six ounces of urine obtained, the specific gravity being 1040. There was a slight

trace of albumin, and sugar at least 5 per cent., by Fehling's rough test. Microscopically, it was negative. During the night there was incontinence of urine and fæces.

On the morning after admission the patient's face was tested for sensation, and the muscles of the left side responded to a pin-prick, but the right side did not respond. Patient is quiet, apparently realizing all that occurs in the wards. He is still unable to open his jaws, except to a limited extent.

November 12th.—It is noted that the right pupil is dilated. At 8 A.M. the patient's vision for form seemed to be abolished. Light perception is still present. Pupillary reaction is good. Otherwise the condition of the man is unchanged. At 9.30 he regained vision for form; paresis of the right eyelid had diminished. At 11 P.M. he was decidedly worse, his pulse being rapid and weak, and respiration accelerated. He became stuporous, and could be aroused.

November 13th.—At 2 A.M. his condition was markedly worse. Pulse was 150, respiration 46, and shallow, with manifest effort. The pupils were contracted, and there was a constant movement of the eyeballs. His head was persistently turned to the left. He swallowed with great difficulty. But slight resistance to movement of the left arm and leg was offered. Later, the patient fell into a state of coma; the skin was dry, mouth partially open, and the tongue apparently in the centre of the mouth. Breathing was not stertorous.

At 2.30–3.30 A.M. he was given hypodermic injections of strychnine sulphate, gr. $\frac{1}{20}$, and atropine sulphate, gr. $\frac{1}{100}$. After this his breathing improved slightly, respiration being fuller. At 7.35 A.M. the patient died.

I was not present at the autopsy on this case, although I had seen the patient during life, and, unfortunately, the exact condition of the cerebral vessels was not carefully noted; but from the location of the softening it was evidently due to occlusion of one or more of the branches of the medicerebral artery. Nothing in the condition of the heart or other organs pointed to embolism, and the strong probabilities are that the case was one of syphilitic thrombosis.

The following are the notes of the autopsy: Post-mortem examination shows the body of a fairly well-developed white male, a little above average height, dark complexioned. Rigor mortis is moderate. The left foot is hyperextended.

Examination of the abdominal cavity shows considerable subcutaneous fat of a pinkish-yellow color. The bladder is filled and extends two and one-half inches above the symphysis pubis. The appendix is normal and lies just above the psoas muscle. Adhesions are found between the liver and diaphragm and between the liver and the transverse colon. The diaphragm extends to the bottom of the fourth rib on the right side, and to the fifth interspace on the left side. The omentum is curled upon itself so as to cover the stomach. The left lung is free but for a tiny adhesion at the apex of the lower lobe, but on the right side there are numerous adhesions posteriorly. The lungs are retracted. Examination of the pericardium shows milk spot on the right ventricle. The right auricle is distended with dark liquid blood. The tricuspid orifice admits three fingers. The left auricle contains a quantity of dark liquid blood. The mitral orifice admits two fingers. The aortic valve is incompetent to the hydrostatic test. The pulmonic valves are normal, and the aortic also, with the exception of some slight fenestrations. The aorta is small in calibre, being 6 cm. in circumference, and is slightly atheromatous. The left lung is rather small. The lower lobe is much congested and slightly œdematous, presenting a condition resembling hypostatic pneumonia. On section

the surface is glistening and congested, and portions sink in water. The right lung has four lobes. The lower part of the upper lobe is very congested, pieces of it sinking in water. The color is dark red, the surface is shiny and slightly granular. There is no increased friability, and a frothy liquid exudes upon pressure. Elsewhere the lung is œdematous. The spleen is much enlarged; it is 16 x 7 x 2 cm., and is soft and flabby. The color is pale red. The left kidney is considerably enlarged. The capsule strips easily, and on section the cortical portion is pale, pinkish-red and marked by red striæ (dilated veins), and slightly reduced in width. The pyramids are somewhat darker. The right kidney presents a like condition. The liver is enlarged. On section it shows an increase of connective tissue about the lobules. The bile-channels are dilated and contain a yellowish-green, opaque, stringy substance. The gall-bladder is not visible, being matted in adhesions between the colon and liver. The pancreas is very firm, and is normal in color and structure.

The scalp presents nothing abnormal. The skull-cap is increased in thickness. The membranes of the brain are much congested, both at the base and convexity. The larger vessels are filled with blood. The circle of Willis is slightly atheromatous. The pia mater over the convexity is slightly opaque.

The brain was opened by Dejerine's method. On cutting down to the level of the third ventricle an extensive area of softening was discovered on the left side, involving the outer part of the caudatum back to the tip of the tail, the internal capsule, and the lenticula. The softening extends to the claustrum, but does not seem to involve it. Internally, it extends to the tænia; posteriorly, to the end of the parietal lobe. The gray matter is pinkish-gray in color. The oblongata, pons and cerebellum presents nothing abnormal. The softened area has a pinkish-white color, white predominating.

The conditions found at the autopsy, summarized, were softening of the striata, congestion of the brain and membranes, hyperplasia of the aorta, hypertrophy of the kidneys, slight parenchymatous nephritis, hypostatic pneumonia, and acute splenic tumor.

The following is a case of softening of the pons due to syphilitic disease of the basilar artery and its branches:

B., aged forty years, was admitted to the Philadelphia Hospital on May 20, 1879. He stated that three weeks before, while lying down, he suddenly felt as if his head was spinning round. He tried to get on his feet, but could not do so on account of the vertigo and a general loss of strength. He did not lose consciousness during the seizure. His speech became so thick that he could not be understood, and he found, on rallying from the first effects of the attack, that he was partially paralyzed in the right arm and leg, and that his face was a little drawn to the left.

Summing up this case the chief points are found to be as follows: headache; a vertiginous seizure, which left partial right hemiplegia; left convergent strabismus; defective articulation; paralysis of the left arm coming on suddenly three weeks after the first attack, but without any special head or general symptoms; a third seizure, accompanied by pallor, anxiety of expression, great emotionality; profuse perspiration, difficulty in breathing and swallowing; inability to speak, to thrust out the tongue or open the mouth widely; paralysis of both arms and both

legs, but more pronounced on the right; convulsive tremor, most marked in right leg; weak and frequent pulse; slight elevation of rectal temperature for four days. On the day of death, deepening of all the symptoms just noted, and, in addition, paralysis of bladder and bowels, conjugate deviation to the right, and rapid elevation of temperature to 108° F. Some interesting negative facts are, absence of anæsthesia, of nausea and vomiting, of true convulsions, and of changes in the urine.

An autopsy was held fourteen hours after death. The pia mater exhibited slight general cloudiness, with scattered patches of extreme redness. On examining the base the basilar artery was found to be enlarged to nearly twice its usual diameter, its walls being thickened and rigid, and rough on the outside. Some of the small vessels which go down from it into the pons could be seen to be obliterated; the basilar trunk itself, however, was quite free and of good calibre, and was readily opened with the scissors. The other main arteries, and their primary and secondary branches, showed widespread evidences of atheroma. The pons was the seat of an extensive and interesting lesion, the following description of which is partly from notes made at the time of the post-mortem examination, and partly from careful subsequent investigation of the specimen. This lesion was an irregular area of softening, the centre of which was just below the centre of the pons. Superficially the softening was an inch in greatest length, which was from above downward and to the left of the median line, and three-fourths of an inch in width at its widest part, which was near its lower boundary. Vertically the softening extended about one-fourth of an inch higher to the left of the median line, while horizontally and below the centre it reached about one-eighth of an inch farther to the right than to the left. It presented four spots of excavation, the intervening spaces being filled up with softened and broken-down tissue; one of these was a comparatively large central space, the second was to the left and above, the third to the left and below, and the fourth to the right and below the centre. The entire surface softening was included in an irregular triangle, formed by joining these outer points of depression or excavation. On subsequent close examination, the central region of the pons was found to be scooped out to the depth of half an inch, and it was also irregularly invaded in all directions by softened tissue; but its upper and lower fifths, and lateral bands from one-fifth to one-sixth of its width, were unaffected. The cranial nerves superficially were not involved. The floor of the fourth ventricle presented a healthy appearance.[1]

Dr. Hoppe[2] reported a case in which the disease was situated in the pons, presenting during life the picture of bulbar paralysis. A rapid left hemiplegia with paralysis of both sixth nerves and paresis of lower face followed by paralysis of all extremities, both facial and hypoglossal nerves, great dyspnœa and rapidity of pulse. Patient died in three days. The lesions affected both sides of the pons, total on the right and partial on the left; also involved both fillets. The paralysis of the cranial nerves was due to the lesion in their paths above their nuclei, these latter (except that of the sensory fifth) being intact.

[1] Mills, C. K.: Brain, January, 1880.
[2] Brit. Med. Journ., April 1, 1892.

The basilar artery was completely obstructed, and a structure resembling a gumma was found in its wall. The patient contracted syphilis ten years previously. He had for some months before his fatal illness suffered from intense and constant headache, generally in the occipital region.

CONVEXITY MENINGITIS AND MENINGO-ENCEPHALITIS.

Focal or circumscribed syphilitic lesions are of more frequent occurrence in certain regions of the base of the brain than in its convexity or any of its lobar surfaces, either of the convexity or of the base. While this is true, focal lesions of the meninges or cerebral surface could not be classed as infrequent. Probably the most common case is to find in connection with well-marked basal syphilis involving large vessels and cranial nerves one or more less important convexity lesions. This combination I have seen somewhat frequently. When a circumscribed area of gummatous meningitis or meningo-encephalitis is present as an isolated lesion, or as one of two or three patches, the most common site of the lesion is the parietal or frontal lobe; and when the latter lobe is the chosen spot, the lesion is quite commonly situated in the posterior half of this lobe. The tip of the frontal lobe and its orbital surface are infrequent sites of focal syphititic lesions, as are also, and even in less degree, the mesal surface of the hemisphere and the occipital and temporal lobes. The motor region is by far the most common site.

Of somewhat frequent occurrence is a diffuse meningitis, which, as it progresses, becomes also a meningo-encephalitis of the brain convexity, or perhaps it would be better to say of the lateral, mesal, and basal lobar surfaces of the cerebrum, excluding the central region of the base. The form of brain syphilis may at first be more or less circumscribed, fusing gradually into contiguous territories, or it may cover a coterminous area of considerable size with additional unconnected spots or areas of disease. The localizing symptoms in such a case have necessarily two or three dominating features. The symptoms which first appear will in time be supplemented by others, indicating extension and dissemination of the pathological process; or under treatment certain symptoms may disappear, while others pointing to new localities may come into the foreground. The most common syndromes are those which point to disease, either of the motor

zone or of the prefrontal region, or of these two combined. These are for the former pareses, paralyses or aphases, and epileptoid attacks, and for the latter, states of somnolence, stupor or apathy, or mental change. Disorders of cutaneous and muscular sensibility and astereognosis, usually of a character not pronounced owing to the slight cortical penetration of the lesion, point to implication of the post-parietal region. Visual aphasias, partial hemianopsias, impairment of word-hearing, and verbal amnesias are sometimes present, and show occipitotemporal involvement. All sorts and conditions of symptoms may appear successively, together, or in almost any fashion as to time of occurrence and order of phenomena in this type of diffuse meningo-encephalitic disease. Optic neuritis may or may not be present; except in a late stage of the disease, it is perhaps oftenest absent.

One of the most striking phenomena of the disease is the almost constant change in the mental status of the individual, a change which is probably due to the dissociation of the cerebral processes by the wide extension of the cortical disease. Headache, sometimes severe, but varying much in intensity in different cases, is a common symptom. This may be more or less localized, and pain on percussion and deep pressure is quite frequently found if sought for by the clinician. The symptoms sometimes resemble those of true cases of general paresis late in the first or early in the second stage, but they may often be made to disappear by energetic antiluetic treatment.

DISSEMINATED ENCEPHALITIS.

The symptoms of local or disseminated encephalitis may be as multiform and irregular as the lesions producing them, and they will often be associated with the symptoms of the associated tumor, meningitis, or arterial disease—indeed, the more pronounced symptomatology of the latter affections will largely mask or overshadow those of encephalitis. Paresis, anæsthesias, amnesias, cranial nerve disorders of slight or marked character, visual, auditory, and other phenomena of the special senses of cerebral origin, may be among the symptoms. Some forms of focal sclerosis of the nerve-centres are due to syphilis. According to Lancereaux, the syphilitic forms of sclerosis can be diagnosticated from the nonsyphilitic by the greater tendency of the neuroglia to fatty degenerations and by the occurrence of foci of softening in their neighborhood. Whether or not true insular sclerosis

of the brain is of syphilitic origin may be regarded as doubtful, although analogy would favor this etiology. Cases of miliary sclerosis and of lobular sclerosis have been recorded with details of autopsies and microscopic examinations of patients with clear histories of syphilis.

CEREBROSPINAL SYPHILIS.

As already stated, my experience, and especially my experience in the Philadelphia Hospital, has led me to the same conviction as that which has been expressed by Oppenheim, Sachs, and others, that syphilis of the nervous system is in one way or another most frequently a cerebrospinal disease, although it often shows a tendency to a well-defined selection of one portion of the cerebrospinal axis for its most decided assaults. While this is true, certain easily recognizable types of cerebrospinal syphilis are frequent. One of the most common of these as seen at the Philadelphia Hospital is that which has been well described by Sachs in a paper in which he gives a valuable post-mortem record, and also a series of almost equally valuable because well-studied clinical cases. In these cases the most common combination of symptoms toward the final stage of the disease is one which shows the existence of diffuse cerebrospinal meningitis, especially leptomeningitis with irregular or widely extended infiltration of the substance of the brain and cord, disease of blood-vessels, large and small, being present in all parts of the neuraxis. The most marked symptoms are those which indicate compression and infiltration of cranial and spinal nerves, infiltration of the white matter of the pons, oblongata and spinal cord, and degeneration of certain regions of the cord, especially the lateral and ventral columns, less frequently the dorsal columns, and still less frequently the gray matter. Cranial and spinal nerve palsies of irregular type, spastic paralysis, mental irritation and apathy, and optic neuritis, are among the most common clinical phenomena.

Both the lesions and the symptoms which are presented by the average case of this type are well summarized by Sachs in his recapitulation of the conditions presented by one of his patients with a good clinical history and an excellent record of autopsy and microscopical examination. This summary is as follows:

"The pathological conditions described above tally well with the clinical history of this case: A married woman who has exhibited

undoubted manifestations of syphilis at different periods in her life is stricken down with headaches, vomiting, and vertigo. Motor symptoms are soon developed, resulting in a complete paraplegia of the lower extremities. From this she recovers to such an extent that she is able to walk about and to leave the hospital. After a few weeks, general symptoms again appear: headache, vertigo, vomiting, and mental dullness; also spastic paralysis of the upper and lower extremities, paralysis of various cranial nerves, and interference with the rectal and vesical reflexes, until coma and death put an end to her sufferings. The special features of this history are:

1. A spastic paralysis of the cerebrospinal type.
2. Recovery from this attack.
3. A second attack of spastic paralysis of upper and lower extremities with cranial nerve involvement.

Comparing the history of this case with the pathological findings, we infer that there was at first a general specific process affecting simultaneously the brain and spinal cord; by antisyphilitic treatment or spontaneously the specific process was recovered from; then the process takes a fresh start, and finally results in special deposits in the pons and cord."[1]

The following cases are good clinical illustrations of cerebrospinal syphilis, and they also illustrate the notable variations which occur in symptomatology during the progress of a case:

D. E., aged forty-three years, a laborer, was admitted to the hospital April 6, 1898. He had had gonorrhœa and chancroids. No history of any secondary eruption was obtainable. His present illness began about a month before admission, when he was seized with severe headache, which was followed by double vision. Both these symptoms have persisted up to the time of report, with occasional attacks of vertigo, but no vomiting. On admission the pupils were slightly unequal, the right being larger. They both responded sluggishly to distance and light. The tongue was clean and normal in appearance. The reflexes were very active. After admission he had several momentary spells of weakness, sometimes nearly falling, but consciousness was not lost.

In June, 1898, the following notes were made: The patient feels dizzy and weak; he staggers when walking, always going toward the left side. He complains of weakness in the left leg. The grip of the left hand is weaker than that of the right. The right pupil is larger than the left, and there is a slight difference noticed between the two sides of the face when the patient laughs or frowns.

Decided weakness is present in the left arm and leg. The reflexes are increased on the left side. The left pupil is dilated. There is a slight incontinence of urine. The patient is restless and fretful, but he has no pain.

[1] Sachs, B.: N. Y. Med. Journ., Sept. 19, 1891.

The eye examination by Dr. de Schweinitz showed that the pupils reacted normally, and that the external muscles moved normally. The arteries were congested and the veins full. Slight perivasculitis and some œdema of the fibrous layer of the retina were observed. In September, 1898, he complained of double vision extending over the last few days. Examination showed some weakness of the left external rectus and diplopia in the left lateral field. Dr. C. A. Oliver examined his eyes and found no gross changes in the eye-grounds. Vision in the right eye was reduced one-third; in the left, one-fourth. Paresis of the left external rectus, with slight paresis of the left inferior oblique, was observed.

On September 28, 1898, his face was drawn slightly to the right, so that he showed his upper teeth. The tongue was protruded straight. All the tendon reflexes were increased, markedly on the left side. No clonus was obtainable. His gait was distinctive only of left-sided weakness. On attempting to walk it was noted that the staggering and tendency to turn and fall toward the left had entirely disappeared. Diplopia had disappeared, although the right pupil was still larger than the left.

On November 10, 1898, the knee-jerks were increased, the left more than the right. Slight ankle-clonus was obtainable on both sides, but no patellar-clonus could be elicited. Biceps-jerks were present, more marked on the left; triceps-jerk was present in the left arm only. Tapping on the left arm, either anteriorly or posteriorly, gave a movement of the wrist. His pupils were equally dilated. No diplopia was present. The grip of his left hand was weaker than that of the right. No ataxia of gait or station was present, and there was no apparent loss of power in the left leg. The tongue protruded straight, but was tremulous. Sensation was good.

The eye examination on November 18, 1898, by Dr. de Schweinitz, showed the pupils to be prompt in reaction, the eye-grounds without pathological lesions, without strabismus or limitations in the movements of the eyes, and apparently without diplopia.

E. D., aged thirty-five years; Scotland; housewife; married; was admitted to the hospital November 1, 1894.

In 1890 the patient began to have headache, which came on every day, usually in the afternoon, and lasted three to four hours. A year later she noticed that her left leg was becoming weak, and trembled when she went up and down stairs. The headache disappeared when the weakness and tremor of the leg began, and she has not had headache since. At the same time she noticed some weakness in the arm. Gradually the left arm and leg became weaker, and she had sensations of tingling and numbness in her fingers, hand, and arm. In about 1892 she began to have vertiginous attacks, in which her sight became dim. At these times she felt confused and tended to fall to the left side. She never completely lost consciousness; had no tinnitus, nausea, nor headache with the attacks. Two years later she began to lose power in her right leg, and very soon was unable to walk, her right arm growing weak at about the same time. Nearly a year later she began to have disturbance of speech, finding it difficult to talk with her usual rapidity, her enunciation becoming slightly staccato or syllabic. She began to see double. Shortly before admission she lost control over her bladder and bowels.

She said she was well until seven years before admission, when she had two miscarriages.

Examination showed her right pupil to be slightly larger than the left; ocular movements did not seem to be impaired. The right side of the face drooped

slightly; there was little better control of the left than of the right side of the face. The tongue movements were normal. The left arm was almost completely paralyzed. She could retract the arm slightly by aid of some of the trunk muscles. She could partially supinate the hand; the thumb was drawn over the palm of the hand and the fingers were contractured at the second phalanges. She had sensations of tingling in the fingers of the left hand, but touch, pain, and temperature senses were preserved, The left leg was totally paralyzed; there was contracture of the ankle at about 65°; the foot was slightly inverted. No loss of sensation could be determined in the lower extremities. In the left leg muscle-jerk and knee-jerk were increased. Front tap, ankle-clonus, and toe-jerk were present. The right leg was paralyzed, but not so completely as the left. She could not flex the thigh on the pelvis; could not cross the right leg over the left. She could partially flex and extend the leg at the knee, but could not dorsal flex foot, abduct or adduct it, nor elevate the heel; she had a slight degree of plantar flexion. She could elevate the right arm to the level of the shoulder, and could flex and extend it at the elbow. The movements from the wrist to the shoulder showed deficiency and weakness. Hand and finger movements were much affected, the hand showing a tendency to assume the same form of flexure which was pronounced on the other side. She could not fully extend her fingers. She was progressively losing power and acquiring conditions of contracture on the right side of the same character as those on the left. The tendon and muscle phenomena were the same on the right and left sides. Toe-jerk was not present on the right; muscle-jerk and knee-jerk were increased and ankle-clonus was elicited.

The muscles in both upper and lower extremities responded to the faradic current. In the left ear the tick of a watch could not be heard; in the right ear it could be heard at a distance of one foot. Taste was normal; smell was retained. Vision, roughly tested, was fairly well preserved; it was distinctly better in the right than in the left. She swallowed water with difficulty. Sensations on the trunk were present.

L. F., aged thirty years, a machinist. On admission to the hospital he gave a history of chancre eight years ago, with bulbo-mucous patches, papular eruptions on his chest. He had nasal catarrh for years. His illness began a year before admission, when he had pain and a sensation of numbness in the right side of his face, with failing vision in the right eye, followed by pain in the right side of the cranium. This lasted for three months and was followed by anæsthesia of the right side of the face. Eleven weeks before he entered the hospital he awoke at night with paralysis of both arms and of the larynx. After two weeks he recovered power in his arms, with improvement of the larynx. Three days later he noticed a weakness in his left leg, which steadily increased until motor power was lost. Later, sensory symptoms appeared in his right leg, with increasing weakness. He lost control of the bladder and rectum. He had severe pain between the shoulders, this being followed by muscular rigidity. He had a severe cystitis.

Examination showed a young man, poorly nourished and much emaciated. When open, his mouth was seen to be drawn to the right, although this was not noticeable when it was closed. His tongue deviated to the right. Motor power in the left leg was entirely gone, and there was marked foot-drop on the left side. The power of flexing both leg and thigh was retained on the right side. The knee-jerks and biceps-jerks were increased, more markedly on the left side. Clonus was decided on the left, but was less marked on the right. Patellar-clonus

was present on the left side and absent on the right, while patellar-reflex was present on the right and absent on the left. Areas of delayed sensation were present on his thighs, and tactile sense was delayed in both lower extremities; in some places the latter was entirely absent. Temperature sense, with the exception of one or two small areas, was lost from the knee down, but was present on the inner side of the left thigh; sensation to pain and touch was decreased. Temperature sense was absent on the right thigh. The anterior right side of his forehead, right temporal region, right side of his face, eyeball on the right side, right side of his tongue, soft palate, gums, and buccal surface of his tongue were anæsthetic, or at least had delayed sensation.

After his admission, girdle pain, starting on both sides of the spine and radiating around the body between the pelvis and umbilicus, developed. Both legs were at times violently flexed by involuntary muscular contractions. Considerable muscular irritability was present in his legs. Bedsores developed. Partial paralysis of the great toe of his right foot came on. He had loss of taste on the anterior two-thirds of his tongue on the right side. Hearing was the same on both sides. Smell had been destroyed by the catarrhal condition.

Later, his legs were again tested for sensation. On the left leg there were areas of hyperæsthesia; a pin-prick felt like a knife thrust through the leg. The left tibia was excessively tender, the slightest touch causing severe pain. Temperature sense on both legs was much confused, and also on the left anterior side of the chest. Hyperæsthetic areas to pain and temperature were present on the left leg. Unilateral sweating involving the anterior half of the right side of the scalp and the upper third of the face was observed. This gradually extended downward and involved the right side of the neck and the anterior part of the right chest as low as the sixth rib.

Examination later showed the knee-jerk on the right side to be much exaggerated; on the left it was absent. Elbow- and chin-jerks were greatly increased. The left leg was contracted, and the patient began to have frequent severe pains in the legs. Ulcers appeared on the top of his head.

Examination by Dr. Mills elicited the following: The motor branch of the fifth nerve on the right side was involved. By placing the fingers over the temporal and masseter muscles on the right side, and having the patient open and shut his mouth, almost total loss of motion was demonstrated. Tendon-reflexes and muscle-jerks in both upper and lower extremities were still greatly exaggerated. Chin-jerk was present. Patellar-clonus was marked. Ankle-clonus was present on the left side, but not on the right. Areas of sensory disturbance remained much the same. He now had control over the bowels, but incontinence of urine continued. He passed enormous quantities of urine. Foot-drop continued on the left side, but motion in the left leg had to a great extent returned.

The eye examination by Dr. G. E. de Schweinitz showed the cornea to be crossed diagonally by an irregular band of infiltration, more dense in the centre, the result of a former keratitis. The eye-ground was not visible, as a scar crossed the pupil. The iris moved freely. The anterior chamber was of normal depth. No abnormalities were observed in the left eye. The disc and arteries were normal.

In the median line, and in the right field of fixation there was homonymous lateral diplopia, due to paresis of the right external rectus. The excursion of the eyeball was perfect in all directions. Diagnosis: right corneal macula, the result of former neuroparalytic keratitis; paresis of the right external rectus.

UNUSUAL SYNDROMES FROM SPINAL OR CEREBROSPINAL SYPHILIS.

It follows from what has already been said regarding the multiple and heterogeneous character of the lesions of spinal and cerebrospinal syphilis, that in many cases unusual syndromes will be presented and that cases will be seen that cannot with accuracy or exactness be related to any of the forms of spinal or cerebrospinal syphilis already differentiated. These cases may have some of the features of any form—of cervical paraplegia, of acute paraplegia, of Erb's chronic spinal paralysis, of a limited or diffuse meningitis or meningomyelitis, or of isolated gummata or of a gummatous meningitis; but the order of onset and the grouping of symptoms in the final period or at different stages may be such as to present an unusual or even a bizarre clinical picture. Occasionally these unusual or aberrant forms of nervous syphilis more or less closely resemble well-known clinical types. Williamson,[1] in a chapter on "Anomalous Forms of Spinal Syphilis," collects such cases into ten forms, as follows:

(1) Brown-Séquard paralysis (hemiparaplegia). Unilateral lesion of the cord; gummatous meningomyelitis.

(2) Triplegia (both legs and one arm paralyzed—hemiplegia and paraplegia). Combined unilateral cerebral lesion, with bilateral spinal lesion.

(3) Cases simulating disseminated sclerosis. Multiple syphiliic lesions.

(4) Cases simulating primary lateral sclerosis. Sclerosis of crossed pyramidal tracts of the spinal cord with slight changes elsewhere.

(5) Cases simulating anterior poliomyelitis. (?) Probably lesion of, or in distribution of, central arteries of spinal cord—anterior median arteries.

(6) Cases simulating amyotrophic lateral sclerosis. (?) Meningomyelitis in lower cervical region.

(7) Cases simulating pseudohypertrophic paralysis and idiopathic muscular atrophy in the gait and manner of rising into the erect position. (?) Meningitis at the lower part of the cord.

(8) Cases simulating syringomyelia as regards sensory symptoms. Meningomyelitis.

[1] Syphilitic Disease of the Spinal Cord, Manchester, 1899.

(9) Cases presenting symptoms of meningomyelitis during life. Pathologically simulating syringomyelia; meningomyelitis with cavities in the gray matter of the cord.

(10) Cases simulating locomotor ataxia (syphilitic pseudotabes). Meningomyelitis invading the posterior columns; gummatous infiltration of the posterior columns.

D. B., aged forty-three years; white; single; cigar dealer; was admitted to the Philadelphia Hospital May 31, 1900.

Ten years ago he had gonorrhœa and two weeks later seven chancres appeared on the penis. He had been exposed to infection between the appearance of the gonorrhœa and the chancres. A short time afterward he had a sore throat but this soon got well. From description it seems to have been follicular tonsilitis. He never noticed any secondary specific symptoms. About a year after the chancres appeared he complained of pain in the left loin, which came on suddenly and was severe. It became worse when he lay down, and after a time he was unable to lie down at all for many days. When this pain improved his sight suddenly failed and his left eyelid began to droop. Both of these conditions cleared up partially under treatment. Shortly after this (in 1893) he attempted sexual intercourse, but failed on account of the erection being transitory and disappearing almost at once. In 1893, for a couple of months he had severe attacks of headache daily.

In December, 1896, on attempting to rise one morning he complained of a quivering sensation in the left side of the upper lip, and found that his left arm and leg were very weak. He had no pain. His left leg became so weak that he could not walk. After four months' treatment in a hospital he recovered the entire use of his arm and sufficient power in his leg to allow him to walk, although he still limped.

Fourteen months ago he noticed on rising in the morning that both legs were weak and paretic. He could just manage to walk with help. He complained also of numbness and pain in the thumb and the three adjacent fingers of the left hand. Since then he has had occasional sharp, shooting pains in the legs. For six months he has been unable to hold his urine and to evacuate the bowels naturally. For some time prior to this he would evacuate the bowels involuntarily.

The last set of symptoms enumerated above has persisted up to his admission to the hospital. An ophthalmological examination, made by Dr. Hansell on June 5, 1900, gives the following results: Right eye, $\frac{15}{20}$; low grade of optic neuritis. Left eye, $\frac{15}{30}$; commencing atrophy following neuritis. Functional divergence from weakness of convergence.

The following notes were made by Dr. Burr on June 8, 1900: The patient is bedridden; numbness in the thumb, index and middle fingers of the left hand and on the ulnar side of the left arm; no numbness of the right hand; both legs somewhat numb; can raise the left leg slightly from the bed; can bend the knee, move the toes, flex and extend the thigh, but all the movements are weak. He can move the toes of the right foot, but only when he moves those of the left. He cannot flex the right knee unless he first flexes the left. He can abduct but cannot adduct the right thigh, which is somewhat smaller than the left; the right calf is also smaller than the left calf. The tissue around the left patella is slightly swollen. The right knee-jerk is absent; left knee-jerk quite marked; no clonus

on the right; on the left a few irregular movements. On stroking the right sole there is marked extension of all the toes, flexion of the knee, dorsal flexion of the ankle. The posterior tendons of the right knee react on percussion, as do those on the outer side. On the inner and outer sides of the left knee they are very marked. Cremasteric-jerk is absent on both sides. There is no difficulty in moving the right hand; the right grip is 45, the left grip 35. All movements in the hands are present; there is no ataxia nor tremor of the hands. No wasting of the tongue is present. He is obliged to urinate as soon as the desire comes. His bowels move only when medicine is given. Sensation on the arms and legs is impaired, the patient not being able to distinguish between a sharp and a dull point. No trophic changes of bones or joints are present except perhaps a beginning Charcot joint of the left knee. Some muscular wasting is present in both legs, although it is not marked. He is not able to distinguish between hot and cold on either leg; on the abdomen he makes frequent mistakes; on the chest and on the arms he answers correctly. The pain-sense is decreased in both legs. His mental condition is good. There is no difficulty in swallowing, but slight occasional difficulty in articulation.

Examination of his eyes on June 9, 1900, showed the pupils to be generally irregular in size, the left being the larger. They react to light and accommodation. The left eye moves to the right well, following the right in observing the point of fixation, but in turning to the left it does not follow so well. He complains of seeing double in certain positions and of weakness of sight of the left eye. He complains of being unable to turn himself in bed.

The patient was kept on mixed treatment in large doses until August 30, 1900, when the treatment was discontinued on account of an iodide eruption on the skin. He says that the pain in his legs and in the thumb and fingers of the left hand are worse than when he was admitted. He says also that his legs are weaker. He complains constantly of a tight sensation in the epigastrium, but says that he has no sense of constriction about the waist. He says that he has been troubled with the epigastric uneasiness for two years, but that it is getting worse. There is no abdominal tenderness.

In September, 1900, the conditions were generally about the same as noted in June.

P. D., single, laborer. He had gonorrhœa when eighteen years old. He has used large quantities of tobacco.

In 1889 he began to have a tired feeling from his waist down, usually after a day's work. This continued for about six months, when intense cramps in the muscles of his legs were present, especially at night. The attacks came on suddenly and were so severe that he would at times jump from his bed with pain. This condition lasted about six months (about a year after the beginning of the trouble). Muscular twitchings were a pronounced symptom. Up to that time he had been able to walk without a cane. In 1890 his condition had become so bad that he had to use a cane. When he tried to walk the left foot struck the right. About October, 1890, he had to give up work, and shortly afterward was taken with grip and pneumonia, followed by pleurisy, which lasted for ten weeks. When he now attempted to walk he found that he could not raise his feet from the ground, and that he had incontinence of urine and fæces. This condition lasted until he was admitted to the Philadelphia Hospital in 1891, and was present at the examination made upon admission. Sight was normal and no palsy was present

in the face or upper extremities. In January, 1900, it was noted that no ataxia was present in the arms. The left leg is one inch shorter than the right; the gait is complicated. His toes drag on the floor. The left leg is seldom flexed, the right constantly. Both knee-jerks are plus; ankle-clonus is present on the right, absent on the left. Babinski reflex is present on both sides. Sensation is normal.

On September 15, 1900, the conditions were practically the same as above noted, except that ankle-clonus was persistent on both sides.

A. T., white, married, has used alcohol moderately and tobacco to excess, and about twelve years ago had gonorrhœa. In November, 1893, he noticed that he would become dizzy and stagger at times, so that it was necessary for him to stand still or sit down for a few minutes. He passed large quantities of urine, especially at night. He did not have at this time any sharp pains in the legs. In January, 1894, on account of increased dizziness and pains in the legs and back, he was compelled to leave work. He became gradually worse, was unable to walk, and was practically bedridden until June 24, 1894, when he was taken to the Presbyterian Hospital. He was a patient there until July, 1895. When taken there he was able to go about at times with the aid of canes. The pains were not so sharp, but he would at times have a dizzy spell, during which it would be necessary to support himself to keep from falling.

He went to the Philadelphia Hospital August, 1895, in about the same condition as above noted. He continued to have an occasional dizzy spell, and still complains of a dull, boring pain in both legs and in the back.

On September 8, 1899, the following notes were made: Thighs and legs well nourished, arms not in the same proportion, while the hands show marked evidences of wasting, especially the intrinsic muscles of the right hand. Station with the eyes open is fair, but he falls when the eyes are closed for a few seconds. In walking he swings the left leg out and drags the toe in a peculiar manner. He needs a cane to walk. Both knee-jerks plus; ankle-clonus slight but persistent; slight plantar reflexes. Sensation to touch, pain and temperature is normal; he has girdle sensation at umbilicus. Examinations subsequent to the above gave practically the same results.

C. H. W., aged thirty-seven years; white; single; machinist; for four years painter by occupation; was admitted to the Philadelphia Hospital September 11, 1900. On admission he complained chiefly of some loss of power in the left arm and leg, which interfered with walking. He also suffered with sharp pains at the nape of the neck.

He had gonorrhœa and a chancre in the autumn of 1892, which seemed to have disappeared rapidly under treatment. He has used alcohol to excess for about fifteen years. Several weeks later a flat pink eruption appeared on the trunk and extremities; he suffered with shooting pains in the legs at night, with sore throat, sores on the gums, and falling of the hair. On the shins and elbows ulcers appeared, which persisted after the other symptoms had cleared up. In December, 1893, he began to have shooting pains at the nape of the neck and in his arms and legs. At the same time he noticed weakness of the left arm and left leg; these extremities began to show contractures. At the end of three months the patient could not move his left arm except at the shoulder; the fingers were clenched and immovable, and the left elbow was rigidly flexed so that the hand touched the collar-bone. This condition was at its height in March, 1894. He could walk with a cane by swinging the left leg at the hip. He was put on specific treatment, and in less

than a month his symptoms had entirely cleared up, paralysis, contracture and pains disappearing.

The patient then remained apparently well until June of the present year (1900), when he began to have dull, aching pain in the head, with drowsiness. His appetite became poor. In July he began to notice weakness of the left arm and leg, which has been gradually increasing, the affected limbs becoming flaccid and showing no contractures. For the last three months he has complained of a dull pain at the nape of the neck. Two weeks before admission (September 11, 1900) he began to have involuntary evacuations of the bladder without his knowledge, this occurring several times in twenty-four hours, although he would urinate voluntarily at the usual intervals. This condition lasted a week, and during the week the patient was troubled with involuntary defecation but once.

He is a moderately nourished white male. The left palpebral fissure is wider than the right. The left side of the face shows the remains of an old peripheral palsy. The left arm is weaker than the right. The patient is able to execute all possible movements with the left leg and left arm, but the movements are slow and awkward. There seems to be some wasting of the muscles of the arm and scapular region. The tongue is protruded toward the left side. The left pupil is larger than the right. Sight in the left eye is impaired. He sees double. The pupils react to accommodation.

The reflexes are as follows: On the right, knee-jerk increased; ankle-clonus absent; Babinski reflex absent; cremasteric reflex absent; bicipital reflex normal. On the left, knee-jerk spastic; ankle-clonus doubtfully present; Babinski reflex present; cremasteric reflex present; bicipital reflex spastic. Sensation to pain and touch is intact. The patient's gait is unsteady and he drags his left leg. He sways slightly more than normal, but is able to stand alone with eyes closed.

The patient under mercurial inunctions and potassium iodide, twenty-five grains three times daily, has become less drowsy, and the pain in the head and neck is not so marked, but there is no perceptible improvement in the arm and leg. These are still weak and the patient limps in walking.

The following case recently reported by Bayet[1] shows an unusual form of alternate paralysis from syphilis. The symptom-complex was probably due to lesions both at the base of the brain and in the upper part of the cord:

"A healthy man, aged fifty-two years, contracted a chancre of the lower lip. Treatment began three months later (when there was roseola), consisted of perchloride of mercury in pill, one-half grain a day. While apparently progressing favorably he began to suffer from severe headache and muscular pains, suggesting influenza, and the left side of his face became paralyzed. There was also paralysis of the right arm with loss of common and thermic sensibility. Taste was absent on both sides of the mouth; vision was impaired on the left side; and the left external rectus was affected, producing diplopia. The left disc was congested and the field of vision was contracted for all colors. The knee-jerks were exaggerated on both sides; the pharyngeal reflex was absent. The secretion of saliva was entirely abolished. The eruption was still present in all its intensity.

"The patient was ordered thirty grains of iodide of potassium daily with mercurial

[1] Journ. de Méd. de Paris, p. 388, Sept. 24 1900.

frictions, and improved very rapidly, so that in thirteen days all paralytic symptoms save the affection of the left external rectus had disappeared, and the eruption was fading."

All these neatly classified anomalous "forms" of spinal syphilis after all simply represent different localizations, extensions and intensities of the syphilitic lesions which are present in those varieties of spinal syphilis which present more distinct and regular syndromes. The more or less irregular Brown-Séquard type of paralysis is produced usually by a peculiarly situated gumma or gummatous meningitis; triplegia by the combination of some well-known syphilitic lesion which ordinarily produces a monoplegia or hemiplegia with a meningomyelitis which uncombined would have resulted in an acute or chronic spastic paraplegia; cases simulating disseminated sclerosis are due to a multiplication of well-known syphilitic focal lesions of small size; cases simulating lateral sclerosis, when clearly syphilitic in origin, are or may be due to a myelitis or meningomyelits chiefly affecting the thoracic region, or are in some cases instances of amyotrophic lateral sclerosis; cases presenting the symptoms of ordinary anterior poliomyelitis are poliomyelitis of syphilitic origin; cases simulating forms of muscular atrophy are inflammatory rather than primarily degenerative; extended or separated cavities, instead of being dependent upon gliomatosis or congenital defect, are the result of meningeal and vascular disease producing necrobiosis; finally, gummatous meningitis may attack the equinal region of the cord, and root-neuritis is, of course, simply a form of syphilitic nerve-inflammation.

In a word, it is sufficient to say that the well-known arterial, venous, meningeal and myelitic lesions of syphilis may attack the cerebrospinal axis in any place, and in any manner in which it is possible for a syphilitic lesion to exist.

The more I see of cases at the Philadelphia Hospital the more I am convinced that, while so-called clinical types like the paraplegia of acute onset and the chronic spinal paralysis of Erb, and special forms of meningitis and meningomyelitis may be sufficiently distinct in their clinical features to allow them to be arranged into varieties, syphilis may so attack the cord as to make it difficult at different periods in the history of a single case to classify these cases clinically under any single head. The chronic spinal paralysis of Erb may have acute

episodes, an acute paraplegia such as has been so well described by Sottas and others may, after partial recovery, be supplemented by features which indicate that the lesions have slowly extended and spread so as to make the manifestations resemble those of Erb's so-called type. The symptoms of a gumma or of several gummata may be preceded or followed by those of a diffuse meningomyelitis, and all sorts of special localizations may be imposed upon any sort of a diffuse or disseminated sclerotic disease. In a single case followed at the Philadelphia Hospital over a number of years I have observed some of the clinical phenomena of every form of cerebral, spinal, or cerebrospinal syphilis discussed in this paper. Such a case thoroughly studied macroscopically and microscopically furnishes illustrations of every known variety of syphilitic lesion of the nervous system.

FEVER DUE TO SYPHILEMIA

Well-known medical writers—Murchisson,[1] Musser,[2] and others—have called attention to the fact that the presence of syphilis is not infrequently shown by a continuous or intermittent fever which cannot be otherwise accounted for. Cases of this description are quite commonly regarded and treated as suffering from malaria. They refuse to yield to quinine, arsenic, or other sorts of antimalarial treatment, but respond favorably to mercury or the iodides, and especially the latter. In a typical case of this description the fever is due to syphilemia rather than to any focal or diffuse specific lesion, although in some cases these are present. The fever may be present in the absence of any focal lesions. In a considerable percentage of cases of all types of nervous syphilis such fever is present, and can only be explained on the hypothesis of syphilemia. In a case of gumma or gummatous meningitis the disappearance of the growth or the products of inflammation may be more or less co-existent with the substance of the fever, but it is not proof that the latter was due to focal or diffuse lesions, as in such cases the poison is circulating in the blood at the same time that it has focussed some of its virulence upon some part of the nervous axis. In practice, a knowledge of the fact that fever is frequent in late syphilis is particularly important in cases of doubtful diagnosis.

[1] Murchisson on Intermitting Fevers.
[2] Univ. Med. Mag., October, 1892.

SYPHILITIC INSANITIES AND PSEUDO-INSANITIES.

In order to make even a sketch of the subject of syphilis of the nervous system complete, it would be necessary to discuss separately the syphilitic insanities and pseudo-insanities; or perhaps it would be better to say the syphilitic insanities, parasyphilitic insanities, and the counterfeits of the latter. The insane department of the Philadelphia Hospital, supplemented by the wards for nervous diseases and the detention and observation wards where patients of doubful mental state are first placed, afford unusual opportunities for studying these forms of mental disorder, and this paper might be much augmented by the records of cases with and without autopsies taken from my private notes and the books of the hospital. The space at our disposal will not, however, permit this to be done, and I shall therefore close the paper with a few quotations from a recent paper by the author,[1] discussing some of the most important problems connected with the subject of syphilitic insanities:

"Omitting mental affections due to inherited syphilis and syphilophobia (melancholia or mania due to the fear of the consequence of syphilitic infection, this not being in a strict sense a true syphilitic insanity), the most important forms of syphilitic insanity are as follows: (1) Syphilitic mental disorders due to the circulation in the blood of the specific toxin (syphilemia), neither specific lesions with detectable symptoms nor degenerative disease being present; (2) true dementia paralytica, or general paralysis of the insane, in its typical uncomplicated form a parasyphilitic disease, and (3) syphilitic pseudoparesis. While true dementia paralytica is a parasyphilitic progressive degenerative disease, its development is sometimes consecutive to true specific lesions, or specific and degenerative lesions may coincide, facts which make it possible to make somewhat numerous clinical subtypes by special groupings of lesions and symptoms.

"Under the first of the above heads, which may be described in brief as the syphilitic toxemic insanities, are to be placed those forms of profound insanity which are not uncommonly designated as syphilitic melancholia (neurasthenic melancholia, hypochondriac melancholia), syphilitic mania, and syphilitic acute dementia (sometimes designated as apathetic or stuporous insanity due to syphilis). Let me briefly

[1] Philadelphia Monthly Medical Journal, vol. i, February, 1899.

touch upon each of these psychic affections. As leading up to the discussion of syphilitic melancholia of profound type, the occurrence of a true syphilitic neurasthenia, a state of physical and mental exhaustion comparable to that which is often seen outside of the domain of syphilis, must be borne in mind. This is usually most marked in the secondary period of syphilis, or may be observed at any stage. It is not to be confounded either with a disorder of nutrition, which results from the excessive use of antisyphilitic remedies, or with the psychic disorders aroused by apprehension and remorse. Syphilitic melancholia is most frequently observed in the second or in the tertiary stage of syphilis, when some of the specific lesions of the disease may be present in the extrinsic tissues of the nervous system, but these are not of such a character as to produce symptoms of the nature shown. The disease is, in other words, clearly a toxemia, whatever other conditions may be present or imminent. It is characterized by more or less profound depression of personality, by depressive hallucinations, by hypochondriac delusions, or by delusions of suspicion, persecution, poisoning, or of unseen agencies of evil, the patient not infrequently showing marked suicidal tendencies.

"In syphilitic mania the patients have hallucinations and delusions often rapidly changing, with more or less incoherence, the whole associated with a state of mental exaltation which may vary in degree from hypomania to mania of the delirious form.

"Instead of either mania or melancholia, in cases by no means rare, the patient in whose blood the luetic virus is coursing presents an apathetic, torpid, or even a stuporous mental state, which may vary from an intellectual obtuseness so slight as to be scarcely noticeable by a careless observer, to a condition so marked as to give good reason for the designation stuporous insanity or acute dementia due to syphilis, while between these two extremes are to be found almost every grade of apathy or torpor.

"On the negative side the patients set apart as belonging to these three forms of syphilemic insanity show no evidences of true specific lesions, focal or diffuse, or primary degeneration of the noble elements of the nervous system—no spasms, no palsies, no optic neuritis, no ophthalmoplegias, and not necessarily any changes in any of the deep reflexes. Of course, instead of a case the mental symptoms of which are simply the indexes of a pure, uncomplicated toxemia, we may have

cases in which, associated with the toxemia, are the evidences of more or less numerous organic changes.

"It is evident that the insanities and pseudo-insanities due to syphilitic toxemias without recognizable organic lesions constitute a class of cases in which prognosis is relatively good and in which, therefore, treatment should be early and actively employed. Incurable dementia and chronic mania should not be the usual termination of such cases, and these results are sometimes due to the non-recognition of the fact that the melancholia, mania, or stupor is the psychic expression of a virus that only awaits elimination by a skillful and bold therapeutist. The mercurial or the mixed treatment is, in my experience, the most applicable to these cases, and the latter is best carried out by active mercurial inunction with rapidly increasing daily amounts of potassium, sodium or lithium iodide. The constitutional impression of mercury should soon be obtained, and the treatment with iodide may in some instances be carried forward after the most approved method of increasing the daily amount taken. In some cases in which, perhaps, inherited predisposition to mental disorder or constitutional weakness plays an important part, even with the release of the patient from the domination of the specific virus, the mental health is not fully established. The mistake is too often made, however, of treating these cases simply with rest, tonics, nutrients and other ordinary but appropriate measures for neurasthenia, hysteroneurasthenia and melancholia of nonluetic origin.

"Syphilitic pseudoparesis is a disease by far more common than pseudotabes, and it has for its chief lesions pachymeningitis, leptomeningitis, meningo-encephalitis, cranial neuritis, and vascular disease, arterial, venous, and capillary, and not infrequently isolated gummata or a few more or less disseminated gummatous deposits. In addition to these primary lesions of pseudoparesis, sooner or later (and usually soon), secondary softenings and indurations occur in various parts of the brain. In order that pseudoparesis shall exist, multiple specific lesions must be present. An isolated syphilitic neoplasm or several such growths, disease of a single or of several cranial nerves, gummatous meningitis in one or several places, and even widespread vascular disease—any one group of these lesions, or any dual combination of such groups of lesions, will not give us a symptom-picture that deserves the name of syphilitic pseudoparesis; in other words, cerebral syphilis

may exist in several well-known types, and with serious symptoms, and yet the disease may not properly receive this designation. When vessels, membranes, nerves, and above all when the cortex become more or less implicated in an inflammatory disease due to syphilis, that affection will give with more or less verisimilitude the symptoms of degenerative general paralysis.

"Syphilitic pseudoparesis is a disease of good prognosis, indifferently good prognosis, or bad prognosis, according to the stage when it comes into the hands of the therapeutist. Like the rare cases of syphilitic pseudotabes uncomplicated by neuronal degenerations, if recognized very early and vigorously attacked it may yield the most brilliant results. The threatened secondary necroses and degenerations should always be before him and urge him to activity in the use of his measures. If not, and the disease has moderately advanced, the prognosis becomes relatively less favorable. When numerous vessels have closed, when the true nervous elements have disappeared as the result of necrobiosis, or have been transformed through imperfect nutrition into indurated masses of mixed tissues, when membranes have become fibrous and incapable of change, the prognosis can no longer be said to be good, and yet the time for the entire discontinuance of treatment has not arrived. The patient should still be plied with measures intended to relieve recent and active inflammatory processes, to remove recently-formed morbid tissues, and even to increase somewhat the contraction of fibroid masses. A stage of transition, and often one in which the tendency is to halting or regression of the specific lesion, has been reached, and now the remedies used should be given in more moderate doses.

"A useful practical comparison can be made between spinal syphilis and nonpsychic cerebral syphilis, especially in their relations to prognosis and treatment. The lesions of spinal syphilis are osseous, membranous, vascular, or myelic, or combinations of these, the most frequent of all true specific lesions being the vascular and the meningomyelitic. As to onset, development, course, and probable outcome, the vascular and meningomyelitic spinal disorders can be more or less closely paralleled with the syphilitic insanities and pseudo-insanities. One form of acute spinal meningomyelitis usually appears comparatively soon after primary infection, most frequently in about eighteen months or less. The disease runs a rapidly destructive or fatal course

in spite of treatment. The irritative and paretic symptoms in these cases may or may not increase rapidly at first, but in many cases culminate with suddenness and rapidity, complete motor paralysis and high anæsthesia often occurring, the patient soon succumbing. A comparable form of rapid syphilitic pseudoparesis is met with, although it is of rare occurrence. The prognosis in both sets of cases is unfavorable, and it is only in exceptional instances that very active specific medication in the prodromic period saves the spinal case from severe and permanent paralysis or death, and the cerebral case from profound dementia or death. A less explosive, but otherwise similar, form of syphilitic vascular and meningomyelitic disease occurs at almost any period after infection, often after many years. The patient under rest, time, and active treatment, especially under large doses of both mercury and iodide, makes a partial and often a large degree of recovery. In like manner, cases of acute or subacute syphilitic pseudoparesis come on long after infection and largely recover, although the persistent psychic degradation is comparatively greater than the corresponding myelic paralysis. The most common chronic form of spinal meningomyelitis is now well known under such names as Erb's syphilitic spinal paralysis and spinal spastic paraplegia. On the one hand I need only say that I have never seen a case of this disease completely cured or approximately cured; and, on the other hand, I have scarcely ever observed a case that was not benefitted in one or more important respects at some stage of its progress. The parallels that can be drawn between this spinal affection and a common type of chronic pseudoparesis is perhaps closer than in the case of any of the other acute or chronic specific spinal diseases. This chronic cerebral affection which so closely simulates true general paralysis has, like Erb's paralysis, a chronic and often somewhat remitting course, some real and some delusive periods of improvement, and slow progress to a mind-destroying termination. One need not go far to see the reason. In both cases meningeal and vascular alterations of a permanent character take place; fibroid conditions of the membranes with obliteration of fine vessels and consequent necrosed areas are irregularly diffused throughout the encephalon in the one instance, and throughout the spinal cord in the other. Treatment benefits the more recent and the recurring specific lesions, and keeps in check serious general toxemia, but cannot restore destroyed neurons."

PTOSIS OF THE LIVER.

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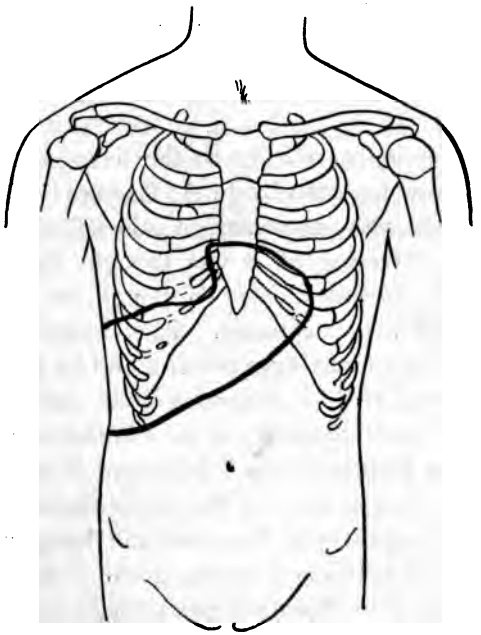
J. B., a white man, aged twenty-nine years, employed as a molder, was admitted to the Philadelphia Hospital on June 28, 1897, complaining of cough, vomiting, weak spells, and swelling of the feet. He dated the beginning of his illness to some three years before, when, while going to work, he was suddenly seized with vertigo. It is said that he frothed at the mouth, but he remained conscious of his surroundings. He had not been quite well from this time, complaining principally of the symptoms named. The feet had begun to swell within a year, and attacks of vertigo recurred from time to time. Vomiting took place after eating. At times there was diffuse tenderness in the epigastrium. Cough had been present for two weeks. The patient had had measles in childhood, but denied all venereal infection. He had been always fairly strong and healthy. He used tobacco moderately, and alcohol not at all. On physical examination the cardiac impulse was seen to be exceedingly diffuse and wavy, being most pronounced below and to the right of the ensiform cartilage. A wavy impulse was visible also over the inner third of the right clavicle. On palpation no thrill could be felt. The area of cardiac percussion-dullness appeared to be merged below with that of the liver. The action of the heart was somewhat arrhythmic, and duplication of the sounds took place occasionally. On auscultation at the apex of the heart the first sound could be heard with clearness and the impact felt with the stethoscope, being followed by a murmur that continued almost up to the succeeding systole. In other situations both sounds of the heart could be heard with distinctness. The murmur was transmitted into the left axilla. No capillary pulsation was made out. A slight difference in the percussion-note beneath the clavicles was appreciable in favor of the left side. The pulmonary percussion-note elsewhere in front was clear, the breathing noisy and accompanied by numerous sibilant râles anteriorly. Posteriorly the percussion-note above the spine of the scapula was less good on the left than on the right. On a level with the spines of the scapula, and in the interscapular region, the resonance was better on the left than on the right over an extent equal to a hand's breadth. Below, the resonance was good on both sides. Râles were pronounced posteriorly, particularly on the left side, and in places partook of the character of coarse crackling. Tubercle-bacilli were not found in the sputum. The area of splenic percussion-dullness appeared not to be increased. The area of hepatic percussion-dullness began on a level with the sixth rib in the right nipple-line, and pursued a horizontal course, with a somewhat downward tendency posteriorly. The lower border ran parallel with, and quite two fingers' breadth below, the

costal arch, extending well into the epigastrium and to the left, merging above with the cardiac dullness. The extent of the liver-dullness, and the relations as viewed from the front, are indicated in the accompanying chart. There is no record of a urinary examination.

The patient remained in the hospital but four days, so that further study was prevented. I looked upon the case as one of displacement of the liver in conjunction with aortic incompetency. There existed also a chronic bronchitis, and the physical evidence pointed to the presence of pleural adhesions. The displacement of the liver could be



Ptosis of the Liver.

explained by the laborious character of the occupation followed by the patient, and it is possible that besides he was accustomed to the use of a belt about the waist, though it is to be regretted that concerning this matter there is no note. This point is of especial interest, as displacements of the abdominal viscera are in general more common in women than in men, and this peculiarity of distribution is attributed to the pressure-differences in the clothing of the two sexes, and especially the use of constrictions by females. Whether or not the existing gastric disorder was in any way related to the displacement of the live

can be a matter for speculation only; but it can easily be understood how such displacement might readily be attended with changes in the relations, not only of the stomach and bowel, but also with such change in the relations of the ducts of the liver and in its blood-supply. The valvular defect probably bore no relation to the hepatic condition. The diastolic murmur, though not heard in the aortic area, not transmitted downward in the course of the sternum, and audible only at the apex, I believed to be due to aortic insufficiency, as there was no evidence of mitral or of tricuspid obstruction or of pulmonary insufficiency, and none of pericarditis. I am unable to explain the anomaly, which is not exceptional in cardiac auscultation. During the short time that the patient was under observation the temperature did not rise above 99°.

The physical conditions relating to the liver in this case are not unlike those in a case reported by F. A. Packard.[1] In this jaundice was present, together with paroxysms of pain referred to the situation of the gall-bladder. During life it was thought that either hepatitis or subdiaphragmatic abscess existed, although, on post-mortem examination, a movable liver was found. Packard refers to several other cases in which like conditions were present, and he dwells particularly upon the possibility of error in diagnosis. Mr. Frederick Treves discusses this subject comprehensively in an admirable paper on "Ptosis of the Liver and the Floating Lobe," published in the *Lancet* for May 12, 1900, p. 1339. He points out that while these abnormalities are common, they are of considerable diagnostic and therapeutic significance.

Ptosis of the liver consists in a sinking down or dropping in the abdominal cavity of the liver, free from gross disease and from other than mechanical causes, such as deformity of the spine or thorax, or pleuritic effusion, or the like. The terms "prolapse" and "dislocation" have also been applied to the same condition. Objection is raised to the qualifications "movable," "wandering" and "floating," as inappropriate. The "floating lobe" consists in a portion of the right lobe, which projects downward in the form of a large tongue-like appendage, and is often associated with some depression of the entire organ, or some tilting down of its right portion.

The liver is suspended in place by its own ligaments, by intra-abdominal tension, possibly by intrahepatic tension, and principally by

[1] Transactions of the College of Physicians of Philadelphia, vol. xviii, p. 230, 1896.

the vena cava. In its descent, therefore, it is the anterior border especially that sinks down, and this may occur in any degree. At the same time the liver becomes flattened and deformed. The displacement is far more common in women than in men, occurring particularly in late middle life. It appears to be due largely to general relaxation of the tissues of the abdomen. It may be associated with movable kidney. The condition may be mistaken for ovarian cyst, cyst or tumor of the mesentery, new-growths of the omentum, hydatid cyst, and, most commonly, for an enlarged and movable kidney.

In the treatment of ptosis of the entire liver a well-made belt, or other form of support, may be of considerable service. Operative treatment has consisted in fixing the organ in place by various means of suture. In the treatment of the floating lobe excision has been practised; or the lobe has been sutured to the anterior abdominal wall; or the distended gall-bladder, with which the condition has often been associated, has been incised and evacuated.

A CASE OF ADIPOSIS.

By AUGUSTUS A. ESHNER, M.D.,

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That increased deposition or increased accumulation of fat is not necessarily or solely dependent upon the quantity and quality of the food is manifested in various ways. It is a not uncommon experience that some individuals will not acquire even a desirable amount of adipose tissue under any or all circumstances, while some become obese in spite of all efforts to the contrary. In these instances heredity appears to exert a more profound influence than the diet. In addition to these conditions of generalized deposition of fat, there may be also localized accumulations, sometimes single, at other times multiple, and occasionally symmetrical. The latter seem to be in no way related to dietetic influences. Finally, the distribution of the fatty tissue may be disseminated rather than either general or circumscribed. In some cases pain is a prominent symptom, and examples of this kind have been grouped together under the designation of dolorose adiposis, as suggested by Dercum.[1]

The first cases of this disorder were observed at the Philadelphia Hospital, and I have myself reported an instance of the kind from the wards of the same institution.[2] I wish, in this connection, to report a case in some respects like the last referred to, but in which pain was not an especially marked feature:

F. S., a woman, aged fifty-nine years, who had been employed as a cook, was admitted to the Philadelphia Hospital complaining of dyspnœa, of pain in the shoulders and knees, and of general obesity. Two years previously there had been shooting pains in the bones, especially in the lower extremities. Shortly afterward the legs began to swell, the swelling at times disappearing, to return subsequently. Swelling of the knees persisted, and was attended with pain, which was aggravated by movement and manipulation. The patient had been increasing in size for several years, and complained of pain in the muscles. On examination she was found to be extremely obese, particularly about the shoulders and the thighs, and the breasts and abdomen were pendulous. The woman was of large stature, and face,

[1] American Journal of the Medical Sciences, p. 521, 1892.

[2] Philadelphia Medical Journal, Oct. 8, 1898; Journal of the American Medical Association, Nov. 12, 1898.

Adiposis. (Present case.)

Adiposis. (Present case.)

Dolorose adiposis. (Earlier case.)

hands, and feet were not disproportionately bulky. She weighed 251 pounds, and was unable to walk unaided. While in the hospital she complained of the right leg feeling cold and the left hot. She suffered a good deal from gastric distress, with nausea and vomiting. The temperature was normal.

The accompanying photographs portray the appearance of the patient, and comparison with the photograph of my earlier case of dolorose adiposis will show the physical resemblance. There is no obvious reason why pain should necessarily be associated with the condition of fatty accumulation, and the case herein reported may be looked upon as belonging to the same class as those of dolorose adiposis, but without marked pain.

The question whether there is any relation between adiposis or obesity and myxœdema is too large and too difficult for consideration in this connection, but it has been shown that thyroid extract, judiciously used, has scarcely a less favorable influence upon the former than upon the latter. This raises the further question, whether abnormal fat-deposition, or fat-accumulation, may not be dependent upon faulty function of the thyroid gland?

In a fatal case of dolorose adiposis, reported by Dercum at the meeting of the American Neurological Association in 1900, the fat, which during life had been supposed from microscopic examination to be embryonal in character, was found to be entirely free from peculiarity. There was, however, interstitial inflammation of the peripheral nerves, degeneration of the columns of Goll in the dorsal and cervical regions of the spinal cord, and unusual pigmentation of the cortical cells of the cerebrum. The brain and the pituitary body likewise presented no alteration. The thyroid gland was small and free from colloid material. The opinion was expressed that the symptoms may have resulted from the presence in the circulation of abnormal substances, resulting from derangement in the function of the thyroid gland. Death had resulted from fatty degeneration of the heart, and there was general fatty infiltration of the internal viscera and the muscles.

At the same meeting Burr referred to a case in which, after death, a new-growth of the pituitary body was found, which, however, was thought to be accidental. There was, besides, distinct disease of the thyroid gland, and interstitial neuritis and degeneration of some fibres of voluntary muscles.

STATISTICS OF TYPHOID FEVER AT THE PHILADELPHIA HOSPITAL FROM JANUARY 1, 1897, TO DECEMBER 31, 1899.

By HERMAN B. ALLYN, M.D.

It has only been since 1897 that a satisfactory method of preserving the histories has been adopted at the hospital. It may seem almost incredible, but is, nevertheless, a fact that the enormous amount of clinical material in the shape of histories of cases at the Philadelphia Hospital has practically been worthless, because, until the past three years, there was no card catalogue of the histories kept, and no index of the cases exists prior to 1897. It is for this reason that the statistics which form the basis of this paper have been limited to the cases which have been dismissed from the hospital or have died in the wards between January 1, 1897, and December 31, 1899.

The whole number of cases in the period mentioned was 184; 135 were males, and 49 females; 146 were white, and 38 black or mulattoes. The fatal cases numbered 31, a mortality of 16.84 per cent. The average age of patients was 26.24 years, the oldest being 53, and the youngest 5; 93 of the 184 being 25 or under, 50.5 per cent.; and 87 being between the ages of 15 and 25 years, inclusive, 47.3 per cent.

The average duration of the fever after admission in cases ending in recovery was 18½ days. The average stay in hospital of the non-fatal cases was about 40 days. This long duration is due to the fact that many of the patients were homeless, and partly to their retention as orderlies when convalescent. In fatal cases the average duration of life was 11.83 days, 14 living only a week or less.

The Fatal Cases.—A study of the fatal cases is always of interest.

1. In one, a woman (A. K., aged thirty-nine years) died on the second day after admission. She had exhibited marked nervous symptoms with nephritis and abdominal tenderness. The autopsy showed nephritis, congestion and œdema of lungs; incipient pericarditis; ruptured ectopic gestation; ovarian cyst and incipient typhoid fever. The death was evidently due to rupture of the Fallopian tube, not to typhoid fever or any of its complications. If this death were excluded, the mortality would be 16.39 per cent.

2. In a second case (L. G. B., colored, aged twenty-five years) the patient was a diplegic from the nervous ward who had high temperature, receiving seventy-one tub-baths in the ten days he lived. The autopsy showed old adhesive pleurisy, hyperplasia of the arterial trunks and typhoid fever.

3. In the third case (J. J., colored, aged nineteen years) the disease seems to have begun abruptly, four days before admission, with chills and high fever. He presented marked evidences of typhoid fever on admission, and subsequently developed nephritis and pneumonia, dying of the latter on the thirteenth day after admission. The temperature kept pretty constantly 103°–104° throughout, except under the immediate influence of baths. It was highest ($105\frac{4}{5}$°) on the tenth day. No autopsy.

4. The fourth case (F. O., aged nineteen years) exhibited marked toxic symptoms from the very beginning of his illness. One week before his admission he had high fever, diarrhœa with incontinence of urine and fæces. The day after admission albumin and casts were found in the urine. The stools continued to be passed involuntarily, even in the tub-baths. Twenty days after admission he was better, passed urine voluntarily, and asked for food. The next day, however, he had a chill, and pneumonia of the left lung developed, the temperature rose to $106\frac{2}{5}$°, the pulse to 144, respiration 40, and he died the following day, cyanosed. No autopsy.

5. In the fifth case (M. D., colored, aged twenty-eight years) the patient succumbed to a complication of pneumonia and nephritis on the forty-fifth day.

6. In the sixth case (A. F., aged thirty-two years) the patient was alcoholic. For the two weeks before admission he had been drinking harder than usual. On the twentieth day after admission he developed a pleuro-pneumonia. The exudates became purulent, and the patient died during aspiration on the forty-first day after admission.

7. In the seventh case (S. S., colored, aged twenty-two years) the patient had been ill three weeks before admission. There was marked toxemia, with delirium, nephritis and high temperature. Four days after admission he had an intestinal hemorrhage of about one pint. The rash was petechial. The next day there was a large hemorrhage from the bowels, and death occurred. Highest temperature, 105° on fourth day. Pulse 88 to 160. No autopsy.

8. In the eighth case (D. P., aged twenty-seven years) the patient was syphilitic. Three weeks before admission the patient had a decided chill, lasting an hour. He had had diarrhœa for two weeks before admission, the stools containing a little blood on the second day before admission. On the day of admission there was an intestinal hemorrhage of a quart. Three days after admission he had severe abdominal pain, and two small, bloody stools. He presented also delirium and tympany. On the fourth day there was vomiting of fecal matter, followed by death on the fifth day.

9. In the ninth case (G. S., aged thirty-four years) the patient developed pneumonia on the fifth day and died the following day.

10. P. L., male; white; aged twenty-four years; Ireland; laborer. Admitted January 11, 1899; died January 14, 1899. Admitted very delirious with a history of several weeks' illness. The tongue was dry, brown, leathery; there were sordes on teeth. The spleen was much enlarged, and there were numerous typhoid spots. Heart's action rapid, sounds weak, pulse feeble. Bronchitis marked. Excessive twitching of tendons. Patient took nourishment very poorly, and had vomiting

and diarrhœa. Widal positive. January 13th, intestinal hemorrhage. Was comatose for twenty-four hours; the pulse was almost imperceptible. Highest temperature 104$\frac{4}{5}$° on day of admission. Pulse ranged from 120 to 160. Duration, two and one-half days. No autopsy.

11. C. D., male; white; aged thirty years; Pennsylvania; cigarmaker. Admitted January 24, 1899; died January 27, 1899. Illness said to date back nine days. Complained of sore throat, weakness and dizziness. Prominent symptoms were loose bowels; abdomen tympanitic, tense and tender; pulse rapid, spleen enlarged. Had two large hemorrhages January 21st, losing about one quart of blood in each. The following morning had another hemorrhage from bowels. Autopsy showed numerous ulcers in large intestine and in lower part of ileum. Lymph-glands enlarged and rather soft.

12. J. McD., white; male; aged forty years; Pennsylvania; laborer. Admitted January 7, 1899; died January 30, 1899. After about two weeks' illness, entered complaining of weakness, headache, and pain in back, legs and feet. Nausea, vomiting, and diarrhœa. Prostration. Abdomen distended and tender. Spots present. Widal positive. January 10th, pneumonia. January 12th, vomited clots of blood. January 16th, left parotiditis; incised January 25th. January 29th, sharp pain with frictions in left mammary region. Died of exhaustion January 30th. Highest temperature 104$\frac{2}{5}$° on third day. Pulse 80 to 120. No autopsy.

13. A. G., white; male; aged seventeen years; Massachusetts; laborer. Admitted December 22, 1898; died December 30, 1898. On admission complained of pain in stomach. His illness began two weeks before admission, with languor, anorexia, headache, nausea, and occasionally vomiting and nose-bleed. Abdomen tympanitic and painful all over. Typhoid spots present. December 24th, delirium, noisy; occasional incontinence of urine; cough. December 27th, Widal positive. December 29th, wild delirium, incontinence of urine and fæces. Urine contained hyaline and granular casts. Died December 30th. Highest temperature, 105$\frac{2}{5}$° on second day; it was frequently 104° or over. No autopsy.

14. P. D., white; male; aged thirty-two years; Canada; weaver. Admitted December 9, 1898; died December 17, 1898. On admission complained of weakness and fever. About two weeks before admission developed headache and vertigo, followed by extreme weakness and diarrhœa. Typhoid spots were present on the thirteenth. The prominent symptoms were delirium, weakness, pulse rapid and feeble, dyspnœa and œdema of lungs. Widal positive. Highest temperature, 105$\frac{2}{5}$° on sixth day. It was also 105° on fifth and eighth days, and as a rule was over 104° in the evening. No autopsy.

15. D. McC., white; male; aged twenty-eight years; Scotland; teamster. Admitted December 12, 1898; died December 24, 1898. On admission complained of pain and soreness over the body. About two months before admission caught cold and had coughed since; had nausea but no vomiting; headache; languor; pneumonia; spleen enlarged; typhoid spots present; Widal positive. Pulse weak, running, dicrotic. Delirium; incontinence of urine and fæces. Diazo positive. Death on the twelfth day. Highest temperature, 103$\frac{4}{5}$° on the third day; pulse, 128 to 140. No autopsy.

16. M. J., white; male; aged thirty-seven years; Pennsylvania; cook. Admitted February 17, 1899; died February 26, 1899. On admission complained of fever, diarrhœa, anorexia, and prostration. Illness began about ten days ago. He took cold, had a number of chills, severe headache, some pain in right iliac

fossa, anorexia, diarrhœa, and bronchitis. Pulse, dicrotic. Heart's action very weak. Additional symptoms were tympany, gurgling, typhoid spots, Widal positive, delirium constant and at times violent, incontinence of urine and fæces, progressive failure of strength and increase of heart weakness. Death on the ninth day. Highest temperature, 104$\frac{4}{5}$° on the first, second, third and fourth days. Pulse, 104 to 130. No autopsy.

17. E. O., white; male; aged twenty-one years; Philadelphia; tinsmith. Admitted January 31, 1899; died February 11, 1899. About three weeks before admission began to suffer with headache, aching over body, languor; three days later became prostrated, and suffered with intense headache, anorexia, vomiting and diarrhœa. Abdomen slightly distended and markedly tympanitic. Additional symptoms were enlarged spleen, typhoid spots, insomnia, incontinence of fæces, tremor of hands and of lower jaw, positive Widal reaction, carphologia, subsultus tendinum, and coma vigil. Subsequently there occurred delirium and suppression of urine. Death on the twelfth day. Highest temperature, 105$\frac{4}{5}$° on the fourth day; it was often over 104°. Pulse, 100 to 128. No autopsy.

18. A. G., white; male; aged thirty-six years; Poland; laborer. Admitted February 8, 1899; died March 2, 1899. On admission complained of cough and pain in the breast. Patient says he had been ill four weeks, beginning with severe headache, followed by cough, pain in the breast, and nausea. Tongue was coated, dry, and fissured. Prominent symptoms were bronchitis, typhoid spots, positive Widal reaction, marked tremor of hands, incontinence of urine and fæces, pulse rapid and weak. Hypodermoclysis was employed, but the patient died exhausted on the twenty-second day.

19. A. L., white; male; aged thirty-eight years; Scotland; teamster. Admitted April 11, 1899; died May 14, 1899. On admission complained of general pain over the abdomen. One week before admission felt very weak, and was obliged to give up work and go home. No headache, but fits of chilliness. Anorexia and loose bowels. Tenderness over abdomen, tongue brown and fissured, sordes on teeth, breath foul. Hebetude. Sleeps with eyes partly opened; muttering delirium. Albuminuria; typhoid spots. Seemed about second week of disease at entrance. Small hemorrhage from bowels on fifth day. From this time until death, which occurred suddenly on the thirty-second day, there was a constant struggle to overcome exhaustion and heart failure. Highest temperature, 104$\frac{2}{5}$° on the second day, 104° twice on third day. Pulse, 90 to 116. No autopsy.

20. M. L., white; female; aged twenty-three years; married; Philadelphia; housewife. Admitted to gynecological wards July 30, 1899; died August 6, 1899. Admitted with a history of abortion on the previous day and with a history of chills and fever which had lasted five days. Uterus curetted and decomposed membrane removed. Spleen enlarged, Widal positive, tympany. Patient transferred to medical wards. Here typhoid spots appeared, the pulse became rapid and weak, the lungs congested, and the patient died soon after a convulsion on the seventh day. Highest temperature, 105° on the fourth day; it was 104° or over thirteen times. Pulse, 96 to 130. No autopsy.

21. J. C., white; female; aged twenty-three years; Germany; domestic. Admitted August 23, 1899; died September 2, 1899. On admission complained of anorexia and pain in back, legs, and abdomen. Illness began two weeks before admission. Prominent symptoms were rapid and dicrotic pulse, arteries sclerosed; typhoid spots, Widal positive, tympany, gurgling. Yellow, liquid stools. De-

lirium supervened and became violent. Occasional incontinence of urine, with albuminuria. Highest temperature, 104⅘° on admission, generally about 102° or under. Pulse, 100 to 144. Duration, nine days. No autopsy.

22. A. M., white; male; aged twenty-seven years; Philadelphia; laborer. Admitted November 30, 1898; died December 16, 1898. On admission complained of cold and sore throat. Often gets drunk on beer. Illness began two weeks before admission with numbness of left side of face and right side of body. He fell to sidewalk and was removed to hospital. Prominent symptoms were hebetude, cough, pain in epigastrium, lips dry and parched, sordes, tongue dry and coated brown. Congestion of lungs, followed by fall of temperature to normal on the fifth day; recurrence of fever on the sixth, with enlarged spleen, typhoid spots, positive Widal, several slight hemorrhages from the bowels, and then a large hemorrhage on the 15th. December 16th, pulse weak, but gained in strength. Respiration frequent and accompanied by bronchial breathing over bases of lungs. Cyanosis. Large liquid stool but no blood. Vomited 1.30 P.M., and died almost instantly. Duration, sixteen days. Highest temperature, 104⅘° on the fourteenth and fifteenth days. Pulse 78 to 124. No autopsy.

23. W. J., black; male; aged twenty-two years; Virginia; laborer. Admitted April 23, 1899; died April 27, 1899. Entered hospital delirious, but with history of illness for five days. Complained of headache, cough, and diarrhœa and epistaxis. Pulse rapid and dicrotic. Bronchitis. Spleen enlarged. Widal positive. Delirium persisted, there was incontinence of urine and fæces, and the pulse became progressively weaker. Death occurred on the fourth day. Highest temperature, 105⅕° twice on third day, and over 104° on three other days. Pulse, 108 to 136; respiration, 32 to 40.

Autopsy.—Typhoid ulcers of small and large bowel. Hyperplasia mesenteric glands, moderate enlargement of spleen; cloudy swelling of heart, liver, kidneys; beginning interstitial nephritis; no cerebral meningitis and no disease of cord.

24. G. T. B., black, male, aged twenty-one years. Admitted December 19, 1898; died December 22, 1898. When admitted the patient was delirious and refused to talk. The abdomen was tympanitic and tender, urine and fæces were passed incontinently, respirations were rapid and shallow, the breath sounds were rough over both lungs, Widal positive. The patient took nourishment poorly and refused to take medicine. He died on the third day. Highest temperature, 105⅘° on the first day. Pulse 128 to 130. No autopsy.

25. J. H., black; male; aged twenty-six years; North Carolina; laborer. Admitted December 10, 1898; died December 23, 1898. Illness apparently dates back four weeks, when he began to sweat profusely, became weak and dizzy, and fell from exhaustion while on the way home. Prominent symptoms, languor, anorexia, headache, enlarged spleen, Widal positive, incontinence of urine, delirium. No plasmodia. Sputum negative. Died on twelfth day. Highest temperature, 105° on fourth and eighth days, 104° first day. No autopsy.

26. E. W., colored; female; aged twenty years; Virginia; housemaid. Admitted January 14, 1899; died January 22, 1899. Complained on admission of headache, fever, and nausea. Nine days before admission began to have vague pains and headache, but kept up until January 7th, when she had to go to bed. Intense headache and nausea. Tongue dry, sordes on lips. Pulse frequent and feeble. Slight bronchitis. Spleen enlarged. Tremor, restlessness, delirium, in-

continence of urine, Widal positive. Highest temperature, 106°, often 105°. Duration, eight days. No autopsy.

27. R. H., black; male; aged twenty-four years; Virginia; laborer. Admitted April 7, 1899; died April 13, 1899. On admission complained of weakness and fever. Illness dates back fifteen days, when he developed headache, chilliness, pains in neck and back; no appetite. Prominent symptoms were drowsiness, hebetude, foul breath, fissured lips, sordes, bronchitis, positive Widal. Patient said to be doing well. Tympanites was noted on the 13th, and he died suddenly. Duration, five days. Highest temperature, 104$\frac{4}{5}$° on fifth day, 104$\frac{3}{5}$° on second day. Pulse 78 to 108. No autopsy.

28. W. E. H., black; male; aged twenty-eight years; Virginia; laborer. Admitted March 23, 1899; died March 29, 1899. On admission complained of headache. Uses tobacco, and alcohol to excess occasionally. Two weeks before admission, began with severe headache and cough. Later, free expectoration, some pain in chest, severe dyspnœa, cardiac palpitation, loss of appetite, nausea and vomiting. Diarrhœa marked. Abdomen distended, tympanitic; gurgling in right iliac fossa. The day after admission he was stuporous, and had incontinence of urine and fæces. Widal positive. On the 27th he had a serious intestinal hemorrhage, and on the 28th and 29th he had a number of hemorrhages from bowels, most of them small in amount. The pulse was very feeble and rapid. Coma, followed by clonic convulsion, closed the scene. Duration, four and one-half days. Highest temperature, 103$\frac{1}{5}$°. Pulse, 100 to 140. No autopsy.

29. L. H., black; male; aged twenty-six years; Virginia. Admitted December 10, 1898; died December 15, 1898. On admission complained of pain in the epigastrium. Illness began about three weeks before admission with chilliness, headache, languor, anorexia, pain in stomach, nose-bleed. Bowels constipated. Fever and delirium at times. Heart weak. Some blood was found in stool on day of admission. The next day there was a bloody stool, and on the 12th a large intestinal hemorrhage. On the 13th there were four hemorrhages, three occurring in an hour and a half. Severe nephritis existed. Patient died on the 15th, being in the hospital four and one-half days. Widal positive. Temperature was low, only once reaching the sponging point. No autopsy.

30. H. B., male; black; aged twenty-five years; North Carolina; laborer. Admitted January 31, 1899; died February 10, 1899. On admission had cough, rapid respiration and pulse, high temperature, pain in the left chest and extreme prostration. Said to have taken grippe one month before admission, but worked until three weeks before admission. Tongue dry and heavily coated, sordes. Rusty sputum. Dullness over left lung, posteriorly and anteriorly, and over right lung anteriorly. Heart rapid and weak. Abdomen tympanitic, and gurgling in right iliac fossa. Muttering delirium. Diarrhœa. Widal positive. February 6th, better, but incontinence of urine and fæces. Subsequently he gradually became weaker and died on the ninth day. Highest temperature, 104° during first four days. Pulse, 100 to 120. No autopsy.

31. M. W., black; female; aged twenty-eight years; Philadelphia; cook. Admitted February 20, 1899; died February 23, 1899. On admission patient too delirious to give an account of her illness. Expression of face vacant, tongue dry and brown, sordes on teeth. Pulse very rapid and small. Lungs show at right base, posteriorly, areas of dullness and crackling râles. Tenderness in splenic area and also in right iliac fossa. Spleen enlarged. Delirium and great restless-

ness. Incontinence of urine and fæces. Vomiting. The abdomen became distended, the breathing labored and gasping, the face pinched, the pulse imperceptible. Death occurred at the end of two and a half days. Highest temperature, 104$\frac{3}{5}$° on second day. Pulse 120 to 160. Respirations, 44 to 76. No autopsy.

The mortality in 184 cases was 16.84 per cent.; or, if we exclude one dying of rupture of the Fallopian tube from ectopic gestation, the mortality is 16.39 per cent. In seeking the causes for such a high mortality, the class of patients forming the great majority of the admissions to the hospital must be borne in mind. They are made up not simply of the poor, but of the homeless, neglected, dissipated, and outcast. Of the last 113 cases, 42 were born outside the limits of the United States and 28 outside the limits of the State of Pennsylvania. Most of the negroes came from other States, particularly Virginia. Moreover, 11 of those born in Pennsylvania were born outside of Philadelphia, so that only 33 of the 113 cases were born in Philadelphia. The significance of this I take to mean that more than two-thirds of the patients were either foreign-born or belonged to the wandering class which is less likely to form fixed home ties, and hence cannot be expected to be so well cared for in sickness. I think it a very fair assumption that the great majority of these patients with typhoid fever never experienced good medical care until they came to the hospital. Again, it is well known that the earlier a patient with typhoid fever comes under proper medical care the better the prognosis, and conversely. Now, more than two-thirds of these patients were admitted in the second week and one-third in the third week or later. Of the fatal cases, 6 were admitted during the first week, 14 after the disease had lasted about two weeks, 7 after it had lasted three weeks or longer, and 2 after four weeks' illness. Three were too delirious on admission to answer questions, and it is assumed that these three were in the second week. In 2 cases the duration of the disease before admission could not be ascertained. Thus, only one-fifth of the cases (6) were admitted during the first week, and 23 after the disease had lasted about two weeks or longer. Delay in sending these patients to the hospital was all the more fatal because they were either ambulatory cases or lacked proper care at their homes or boarding-places.

In Osler's series of 229 cases the mortality was 9.6 per cent. Of the 22 fatal cases, 10 were admitted during the first week, 5 during the second, 3 in the third, and 2 in the fourth. The percentage of

deaths to patients admitted in the first week was 9.5; 6.2 for patients admitted in the second week; 12 per cent. for the third, and 25 per cent of those admitted in the fourth week. The statistics of the German Hospital, as reported by Dr. J. C. Wilson,[1] cover 408 cases up to October 1, 1894, with a mortality of 7.8 per cent. These cases were treated by the Brand method of tub-bathings. In speaking of the contention of Brand that the influence of the treatment upon the mortality is proportionately favorable as it is instituted early in the course of the attack, Dr. Wilson says: "Taking Series 4 and 5 together, we note that of the 32 cases admitted not later than the fifth day, 1, or about 3 per cent., terminated fatally. In 78 cases admitted prior to the tenth day the death-rate was 7.7 per cent., and in 18 cases admitted after the tenth day the death-rate was 22 per cent." As autopsies were obtained in very few of our cases, the causes of death have to be inferred from the clinical histories.

The causes of death in the Philadelphia Hospital cases, following Osler's classification, were as follows:

1. *Asthenia*, a result either of the rapid or slow action of the toxins, or a sequence of the severe diarrhœa, 14.

2. *To intercurrent affections*—in the hospital cases principally to pneumonia, with or without nephritis, and in one case to empyema—8.

3. *To accidents of the lesion*, erosion of a blood-vessel, 5, or perforation, 3.

In one case the cause of death was a rupture of the Fallopian tube from ectopic gestation. There was also pericarditis. It will be noticed that half of the fatal cases occurred in negroes or mulattoes, although they constituted about one-fifth of the whole number of cases. This fact accounts, in part no doubt, both for the mortality and for the relatively large number of deaths from pneumonia; for it is well known that negroes offer less resistence to disease than whites, and that they are especially susceptible to pneumonia. On the whole, when it is borne in mind that the great majority of the patients who come to the Philadelphia Hospital are from among the poorest and most depraved in the city—patients, therefore, in many instances already weakened by bad hygiene, insufficient food, and vicious habits—the results obtained from the treatment and the nursing cannot but be regarded as highly satisfactory. Nevertheless, the mortality from pneumonia as a com-

[1] American Text-book of Applied Therapeutics, p. 240, Philadelphia. 1896.

plication (8 cases) would have been lessened by treating the typhoid fever patients in a ward where no pneumonia cases were admitted. Such separation has not been accomplished yet. In future, also, some of the patients who would otherwise die from hemorrhage or perforation will be rescued by prompt surgical interference.

COMPLICATIONS AND SPECIAL SYMPTOMS.

Chills and Fever.—Chilliness and chilly sensations, of course, occurred in many of the cases (in 30 of the last 113), but in 8 cases there were marked chills, which recurred and were followed by fever, and 3 simulated malarial chills, but no plasmodia were found in the blood. This agrees with Osler's statement that among 685 cases of typhoid in not a single instance were the plasmodia found in the blood during the course of the disease.

Incontinence of Urine and Fæces.—Incontinence of urine and fæces occurred in 21 of the cases ending in recovery, and in 13 of the fatal cases; in 2 there was incontinence of urine alone. In one of the former the fæces were passed while the patient was taking tub-baths. Dr. J. C. Wilson says he has never known an instance in which fecal incontinence occurred during the bath.

Nephritis.—Nephritis occurred in 22 of the cases ending in recovery and in 7 of the fatal cases. Tub-baths do not appear to influence unfavorably the course of the nephritis. In one instance at least in which the urine became scanty and contained casts and considerable albumin, under a continued use of tub-baths, or perhaps one should say in spite of a continued use of the baths, the urine became abundant and the nephritis improved.

Sweating.—Free sweating occurred in 14 of the cases.

Bloody Stools.—Bloody stools occurred in 9 cases ending in recovery, hemorrhage in 7 cases, and, as already mentioned in speaking of the fatal cases, hemorrhage occurred in 6 cases and hemorrhage followed by perforation once.

Diarrhœa.—Twenty of the fatal cases were characterized by diarrhœa, and in 38 of the cases ending in recovery (including the 9 who had incontinence) diarrhœa or loose bowels are mentioned. In many of these cases, however, the diarrhœa was not marked, and in a number of these existed before entrance to hospital, and at the time of entrance, subsiding after the patient had been put to bed and kept on suitable

diet. Diarrhœa certainly cannot be looked upon as characteristic of of the disease as we see it at the present time.

Vomiting.—Vomiting occurred in 9 of the fatal cases.

Tuberculosis.—Three of the patients who recovered were tuberculous before the onset of the typhoid fever, and in one of the fatal cases tuberculosis also existed. There was no autopsy.

Boils.—Boils, I am sure, occurred oftener than the ward notes of the cases would lead one to suppose. In one case numerous boils were succeeded by ulcers which, for the most part, extended through the skin only, but in a few instances through the muscle also, becoming bedsores on the sacrum and hips. They were very sluggish and difficult to heal, owing to the greatly reduced vitality of the patient.

Pregnancy.—One of the patients was pregnant, but there is no record of abortion following, so that the presumption is that pregnancy was not interrupted, which is very unusual. Abortion occurred in three cases.

Orchitis.—Orchitis as a complication occurred in two cases, one of which has been reported by a member of the staff (Dr. A. A. Eshner[1]), who also gives a valuable review of the literature of the subject. In the other case the records, unfortunately, are imperfect, but the orchitis appears to have involved both testicles and to have developed early in the disease. The patient was ill three weeks before admission to the hospital, where he was first assigned to the surgical ward. It may be that the orchitis was in origin independent of the typhoid fever.

Phlebitis.—Phlebitis occurred in 3 cases, but was not especially noteworthy.

Purulent Otitis.—This complication occurred in 15 of the cases, and appears to have been readily amenable to treatment.

Relapse.—Relapses occurred in 8 cases. These, of course, were relapses in the strict sense of the word, not merely recrudescences. One patient had three relapses. This patient developed typhoid fever about fifteen days after confinement in the maternity ward of the hospital. She remained, altogether, four months in the medical ward, and at the end of that time was transferred to the gynecological ward for an Alexander's operation.

The Gruber-Widal serum reaction was reported positive in 95 of the last 113 cases; in 13 it was negative, and in 5 the blood was probably

[1] Philadelphia Medical Journal, May 21, 1898.

not examined. This gives a percentage of 84 in which the serum reaction agreed with the final clinical diagnosis.

Other symptoms which are not common, and which occurred in a few of the last 113 cases are the following: Retention of urine, 5; petechial eruption, 2; convulsions, 3; and cellulitis of scrotum, tender toes (acroparesthesia), pleural effusion, insomnia, glossitis, tonsillitis, parotiditis, in 1 case each.

LARGE ABSCESS OF THE LIVER. ASPIRATION; LATER, OPERATION; RECOVERY.

By DAVID RIESMAN, M.D.,
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Abscess of the liver of the variety of which the present case is an example is rare in this locality, if we may judge from clinical experience and from the records of an institution as large as the Philadelphia Hospital. During a period of three years only two cases were observed, both of them at autopsy; and as in this time more than 25,000 persons entered the hospital, the percentage of cases of abscess of the liver, if my own is included, is only a little over 0.01 per cent. There is a record of one other instance of abscess of the liver, but it was a case of multiple abscesses implicating the biliary channels, and it does not belong to the category with which I am dealing.

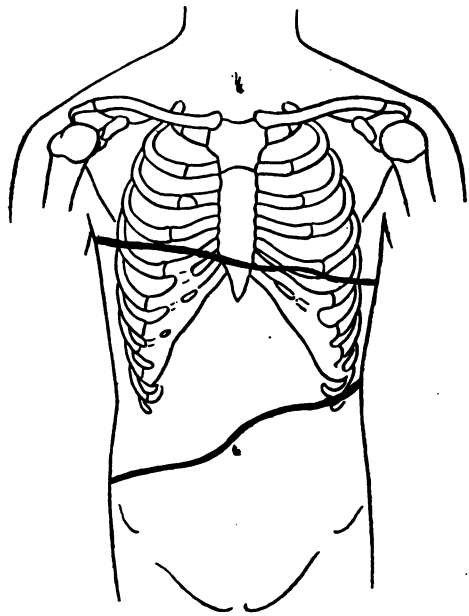
The patient whose case I beg to report is a Belgian, fifty-two years of age, a gardener by occupation.[1] Coming to this country directly from his native land twelve years ago, he has ever since lived in the immediate vicinity of Philadelphia. Both parents died at the age of eighty-six; a sister died of a cause unknown to him. He had small pox and measles in childhood, an obscure fever at the age of twenty-six, and a similar febrile disturbance three years later. Two years ago he suffered from an attack of diarrhœa with hemorrhage from the bowel, but unaccompanied by much pain. He entered the Presbyterian Hospital, and was operated upon for appendicitis in February, 1898, his stay in the hospital extending over a period of sixty-two days. Afterward he was under treatment at the same hospital for cirrhosis of the liver. In March of the present year he had another attack of diarrhœa with bloody stools, again without any considerable pain or tenesmus, and received treatment at the Medico-Chirurgical Hospital. Thereafter he suffered much from pain in the right hypochondrium, and for the relief of it entered the Philadelphia Hospital on June 14, 1900.

He was a well-nourished man, but appeared much older than his actual age. His color was good and the mucous membranes healthy. His chief complaint was pain in the right hypochondrium, most severe when lying down or sitting upright, least severe when semi-recumbent. Lying on the left side was difficult and increased the pain materially. The chest was very broad at its lower part, and had

[1] I am indebted to Dr. F. T. Woodbury, formerly Resident Physician of the Philadelphia Hospital, and now with the Volunteer Army in the Philippines, for the notes of the history.

the appearance of a cylinder flattened antero-posteriorly. The left side moved more freely than the right. On palpation the tactile fremitus was found to be diminished on the right from the fourth rib downward. Percussion yielded natural resonance on the left side; on the right, however, dullness began as high as the fourth interspace in the anterior axillary line, and fused below with the liver-dullness. Above the fourth interspace the resonance was somewhat tympanitic. Posteriorly tactile fremitus was absent on the right side from the eighth interspace downward. The breath-sounds and vocal resonance were unobtainable over the dull area on the right side. The abdomen was somewhat prominent. The liver projected far over the left and could not be separated from the spleen by percussion. Its lower border reached at first to about the level of the umbilical

FIG. 1.

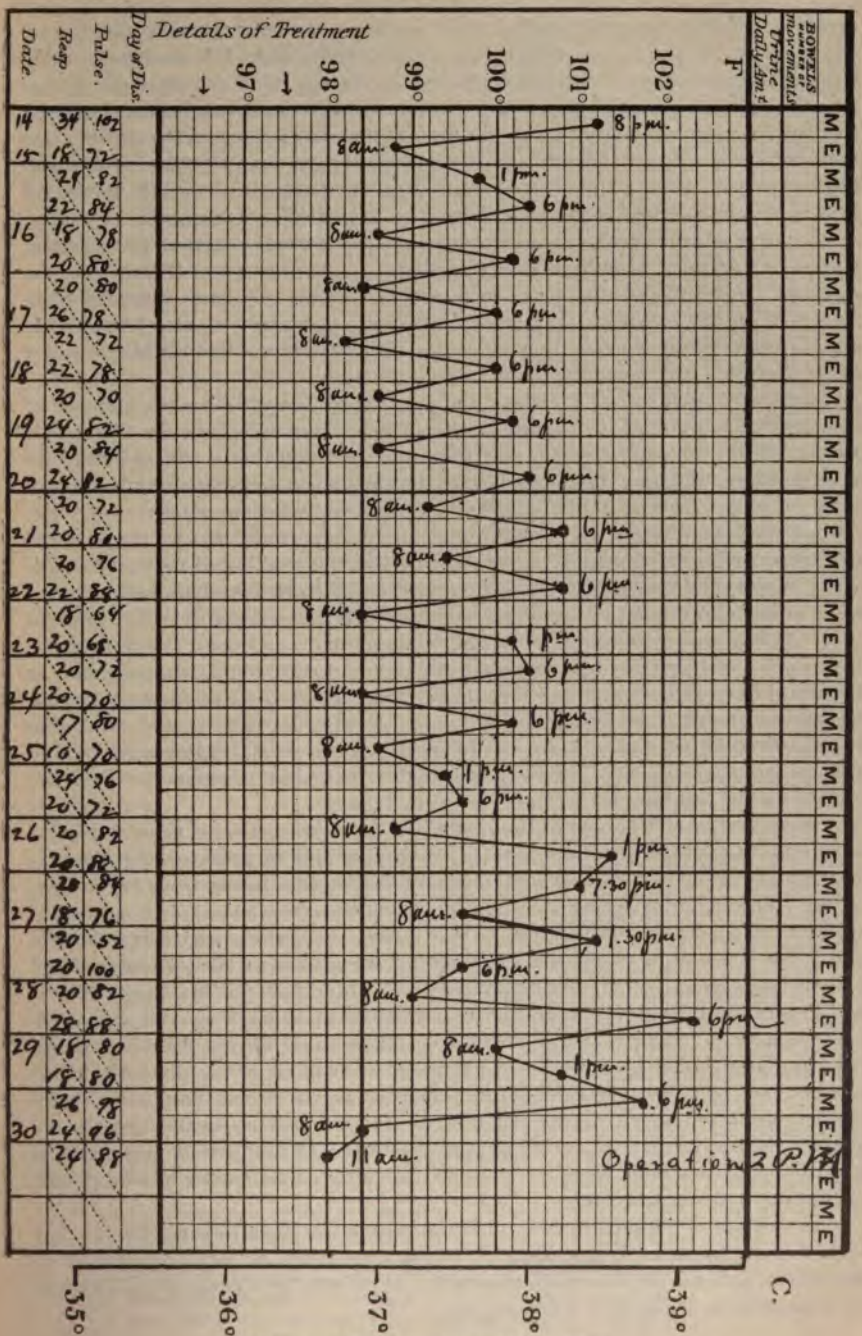


Outline of liver-dullness before operation.

line; later, nearly down to the right anterior superior spine of the ileum. From this point the border extended obliquely upward to the left hypochondrium, just touching the umbilicus (Fig. 1). In the left mammary line it was a little below the costal border. There was some tenderness on pressure over the liver, but it was not marked, and auscultation revealed nothing. The man's temperature, as a rule, ranged from 98.4° to 100.8° (Fig. 2). Only thrice after the day of entrance did it exceed 101°. There were sweats, but no chills, and the pulse was usually between 70 and 80; the respirations were between 18 and 24. The urinary secretion was scanty, but the bowels were freely open. Although the appetite was not good, the man held his own and did not give the impression of being very ill.

Abscess of the liver was suspected, but in view of the history of a previous appendicitis it was thought not impossible that a subdiaphragmatic abscess might

FIG. 2.



Temperature chart. Abscess of the liver.

exist. Aspiration was performed in the seventh interspace in the midaxillary line, and 2537 cc. of fluid were withdrawn. After the tapping the man was greatly relieved; the sweating particularly was less annoying. The day after the operation the dullness extended from the fifth rib above to a point two inches above the level of the anterior superior spine in the right mammary line; in the median line the lower border was at the umbilicus. Posteriorly the upper border of dullness had descended one interspace, and was now in the ninth. In the left mammary line the lower border of the liver was one inch below the costal margin.

Two days later the patient's symptoms became decidedly aggravated. He had a great deal of pain, and there was pronounced tenderness below the ribs on the right side. The liver-dullness had increased perceptibly, and now extended from the fourth interspace in the anterior axillary line, down almost to the level of the anterior superior spine of the ileum. In the right parasternal line the lower border of the liver was just below the umbilicus.

As the rapid refilling of the abscess rendered it unlikely that mere aspiration of the contents would lead to permanent cure, I asked my colleague, Dr. Charles H. Frazier, to see the patient. He concurred in the opinion that a radical procedure was necessary, and accordingly performed hepatotomy in a most skillful manner on June 30, 1900. He has kindly furnished me with the following notes:

"It was evident from its situation that the abscess could best be reached and evacuated by the transpleural, rather than by the transperitoneal route. Accordingly, a portion of the eighth rib was resected and the costal and parietal layers of the pleura sutured together, in order to wall off the pleural cavity. The parietal layer of the pleura was very adherent to the diaphragm, and the diaphragm to the liver itself. Both pleura and diaphragm were very much thicker than normal, as the result of an intense inflammatory infiltration. From the abscess cavity, which was quite superficial, about three quarts of a muddy, chocolate-colored fluid were evacuated, after which the cavity was irrigated with a normal saline solution and tubular drainage inserted. It was estimated at the time of operation that the abscess cavity was from six to eight inches in diameter.

"*Post-Operative Notes.*—For the first week the discharge from the abscess was so profuse that it saturated two or three thick dressings in the course of twenty-four hours. The area of liver-dullness diminished rapidly in size from day to day; on the tenth day after operation it was noted that the upper margin had receded to the sixth rib, and the lower margin to two inches below the costal margin of the ribs. For a few days immediately succeeding the operation the patient suffered pain from a cough, which was attributed to the injury to the diaphragm necessarily inflicted during the operative manipulations; apart from this, convalescence was uninterrupted, and at no time was there cause for the slightest anxiety regarding the patient's recovery. The abscess cavity filled in rapidly with granulation tissue; the area of liver-dullness receded to its normal boundaries; the temperature reached 101° on the fifth day, but from that time on the case ran an almost afebrile course; and the general condition of the patient improved as rapidly as the local condition, so that after the lapse of six weeks he was practically ready to be discharged."

The opening of the pleural cavity naturally gave rise to a pneumothorax; but aside from a transient dyspnœa and a slight cough; the patient experienced no discomfort, and the air was soon absorbed. At present he is entirely well, except for a small sinus at the point of operation.

Description of the Abscess Fluid.—The fluid did not have the characteristic features of pus; it was reddish-brown or chocolate-color, like Anchovy sauce, perfectly opaque, and without much tendency to froth. It had no odor and was not spontaneously coagulable.[1] There was no tendency to separate into layers or to deposit a sediment on standing. When boiled its color changed from a chocolate-brown to a butternut-brown, with a precipitation of albumin to the amount of one-fourth by bulk. The specific gravity was 1037; the reaction neutral. The test for urea by the hypobromid method gave negative results; sugar was also absent.

Microscopic Examination.—Microscopic examination of the fresh fluid showed a great many fat-globules of all sizes; slightly granular, polygonal cells, with eccentrically placed nuclei (liver-cells); and small numbers of red and white corpuscles, the former still possessing their yellowish tinge. Bilirubin crystals, in the shape of small, bright, copper-colored rhombs, were present, and in addition, some peculiar, colorless, elongated, rod-shaped bodies, with pointed ends. The latter looked liked gigantic bacilli, and were motionless, refractive, and generally longer than the diameter of a red corpuscle. These bodies interested me very much, but I was unable to explain their origin until, on looking up some of the descriptions of hepatic abscess fluid, I found bodies described by Netter[2] which seemingly are identical with those observed by me. Netter at first looked upon them as bacteria, but afterward, on further study in other cases, he concluded that they were crystals; indeed, he considers their presence in pus as diagnostic of an hepatic origin. It would be very interesting if this could be substantiated.

Bilirubin crystals, such as were present in the abscess fluid, have been quite frequently found. Renvers[3] described them in 1890. While they may be derived from the bile, it is quite possible that they may come from the hemoglobin of destroyed red corpuscles. In that case they would properly be called hematoidin crystals. It is now generally accepted that, chemically, hematoidin and bilirubin are identical.

Cultures were made from the pus obtained by aspiration and at operation in bouillon and on agar, but the tubes remained sterile; nor were any bacteria found in the pus on staining. Careful search was made for amœbæ, both in the fresh pus and in stained preparations, with entirely negative results. The pus was stained by the method of Mallory,[4] which has proved very satisfactory in my hands in the case of sections from dysenteric intestinal ulcers.

We also tested the digestive properties of the fluid. A small shaving of hard-boiled egg was placed in the native pus; another in pus to which a little alkali had been added. Both preparations were kept in the incubator. The alkali had been added to aid the action of any trypsin that might be present. It has been held by Salkowski[5] and others that the liver cells contain trypsin and invertin, and that these enzymes exist in the cells themselves and are concerned in intracellular digestion. Neumeister,[6] however, believes that the ferments found are nothing more than those that have entered the circulation from the glands and

[1] The fluid was exhibited at a meeting of the Philadelphia Pathological Society.
[2] Netter: Société méd. des hôpitaux de Paris, vol. vii, p. 636, 1890.
[3] Renvers: Berliner klin. Wochenschrift, p. 165, 1890.
[4] Mallory: Journal of Experimental Medicine, vol. ii, p. 529, 1897.
[5] Salkowski: Quoted by Neumeister.
[6] Neumeister: Lehrbuch der physiologischen Chemie, p. 136, 1897.

organs where normally they are manufactured. In both tubes the egg remained unaltered, this proving the absence of any proteolytic ferment.

The pus secured at operation was of the same character as that obtained by aspiration. Although no amœbæ and no bacteria were found, it was still possible that either of them, or both, might be present in some form not recognizable by the ordinary methods of investigation. A considerable quantity of pus was therefore introduced into the rectum of a cat, the animal retaining nearly all of the material. On the following day a diarrhœa developed and continued for several days. It was of a mild type, and the stools contained neither blood nor mucus, and the animal apparently had no pain and ate a fair amount of food. After the diarrhœa had stopped the cat was seemingly as well as ever.

In many respects the pus of hepatic abscess is peculiar; indeed, it usually differs from that formed in all other organs, and even from the pus in cases of biliary or traumatic abscess of the liver itself. Its formation seems to be due to a rapid necrotizing influence on the liver tissue. Manson and Galloway[1] were of the opinion that its formation was the result of necrosis rather than of a pyogenic process. Leucocytes are few in number—a strange feature, as in other forms of pus they are abundant, even when the tissue-cells proper of the organ or part are destroyed by the pyogenic cause. To explain their paucity in hepatic pus, three reasons suggest themselves:

1. Leucocytes may be rapidly destroyed.

2. The blood-vessels may be occluded by thrombi, preventing the emigration of leucocytes.

3. The abscess may contain a negatively chemotactic substance which repels the leucocytes.

The first possibility cannot be excluded, although it seems unlikely that the destruction is so rapid as to annihilate all the leucocytes in a short time. In the abscess fluid procured from the case at operation they were just as scarce as in that obtained a few days before with the aspirator, although the former must have been to a large extent of recent formation.

For the second explanation no confirmatory observations exist; moreover, the presence of red blood-corpuscles in the fluid shows that the circulation in the wall of the abscess was maintained.

Most reasonable is the third explanation, although its correctness is not demonstrable, and its proof can be approached only by means of experiments like those of Lebert, Buchner, and others, on chemotaxis.

From a bacteriologic point of view, hepatic abscess is also very

[1] Manson and Galloway: British Medical Journal, vol. i, p. 676, 1894.

interesting. Though often sterile, it may contain pyogenic organisms or the amœba coli, or both at the same time. Giordano,[1] of Venice, in seventy-two cases found the pus sterile in 58.4 per cent.; in 20.7 per cent. amœboid elements were present, and in 9.6 per cent. bacillus coli and pyogenic organisms were found. Most authorities believe that the sterility is a secondary feature, that bacteria are originally present but suffer destruction, either through the influence of the bile or through some other bactericidal constituent of the abscess fluid. The bile probably has but little to do with the sterility, and the other hypothetic agent is rendered problematic by the experiments of Achard, who was able to cultivate bacteria in the fluid of a sterile hepatic abscess. The sterility of the abscess-contents does not preclude the presence of living bacteria or amœbæ in the abscess walls. The mode of origin of hepatic abscess—usually through the medium of ulcerative processes in the intestine—is strongly in favor of a micro-organismal cause. Perhaps this cause at times is a member of the anaërobic flora of the intestinal tract.

Diagnosis.—The diagnosis of abscess of the liver is, as a rule, quite easy. This is particularly true of cases developing during or immediately after a known attack of dysentery. When, however, the patient is seen long after the subsidence of the dysentery, or when the history of previous disease throws but little light on the case, then the recognition of an hepatic abscess may be a most difficult problem. Under these circumstances, "the secret of a happy diagnosis," to use the words of Patrick Manson,[2] "is to suspect the disease."

Among the symptoms of value in the diagnosis of abscess of the liver—and I refer here to the large abscess only, although the symptoms are quite similar in the smaller multiple abscesses—the following are the most important:

1. *Pain with a sense of weight in the right hypochondrium.* If the abscess develops acutely during an attack of dysentery the pain may set in suddenly, its onset coinciding with the lodgment of the infecting embolus in a branch of the portal vein. Ordinarily the pain is dull, and is either permanently located in the right hypochondrium or radiates into the right shoulder—rarely into the right groin. The

[1] Giordano: Thirteenth International Medical Congress, 1900. Abstract, Münch. med. Wochenschrift, August 28, 1900.

[2] Manson: Quoted by Boinet.

seat and radiation of the pain will depend somewhat upon the site of the abscess—whether it is in the right lobe, where the majority are situated, or in the left lobe; whether in the convexity or nearer the under surface of the organ. Rouis's[1] statistics give the relative frequency of the involvement of the hepatic lobes as follows: Right lobe, 154; left lobe, 33; spigelian lobe, 9.

The patient is usually most comfortable when lying on his back, and as a rule he cannot lie on his left side on account of an increase in the pain and dragging, as William Saunders[2] noted over a century ago. At times the right side is curved in so that the right shoulder is brought nearer to the right hip.

2. *Enlargement of the liver* is one of the most trustworthy signs of abscess, and is almost never absent in the variety with which we are concerned. The liver is enlarged vertically and transversely. In our own case it extended upward to the fourth rib in front, to the eighth rib posteriorly, while its lower border was below the umbilicus. The increase in size is usually rapid—a point of considerable value in the diagnosis. Mensuration will reveal an enlargement of the right side of the thorax. The distention of the side causes a change in the direction of the ribs, which, as Leblond[3] points out, is of value in the differential diagnosis between hepatic abscess and pleural effusion. In the latter the ribs are depressed and their course is more oblique, while in abscess of the liver they either run more horizontally than normally, or their natural obliquity is not accentuated.

3. *The enlarged liver is often tender on palpation*, the tenderness being probably dependent upon an associated perihepatitis. As the latter is sometimes wanting, tenderness is not a constant symptom.

4. *Fever*. In a majority of cases the fever is of the hectic type; and in the presence of such a fever, if malaria and tuberculosis can be excluded, hepatic abscess should be suspected. But there are cases, as our own for instance, in which the fever is moderate—merely irregular, without notable remissions or intermissions, profuse sweats being the only suggestive feature. In probably one-fourth of the cases, fever and sweats are slight or entirely wanting.

5. *The contour of the chest may be altered* in a striking way, as has

[1] Rouis: Recherches sur les suppurations endémiques du foie, Paris, 1869. Quoted by Lancereaux.

[2] Saunders: A Treatise on the Structure, Economy, and Diseases of the Liver, p. 209, 1795.

[3] Leblond: Diagnostic et traitement des abscès du foie, Paris, 1893.

already been mentioned. The chest is very wide transversely and comparatively shallow from front to back, having the appearance of a *flattened cylinder*. The altered shape is due to the pushing out of the lower ribs and a fore-shortening of the liver through a loss of resiliency. If the abscess is small and deep-seated, the change in contour will probably not be produced.

6. *Jaundice* is rare, and occurs only in about one-fourth of the cases. It was absent in my own. According to Lancereaux,[1] icterus and severe vomiting in cases of abscess of the liver point to its situation on the inferior aspect of the organ.

7. *Fluctuation* is not common, but in large abscesses located superficially it may be obtained.

8. Occasionally *œdema* is noticed over the hepatic region. It was not present in our case. Boinet[2] also speaks of a *local elevation of temperature*, which he found particularly in tropical abscesses.

9. Hassler and Boisson,[3] in 1896, called attention to a deep *ballottement* and a firm, *elastic resistance* revealed on percussion and palpation of the liver; and, furthermore, pointed out the presence on auscultation of a fine respiratory *crepitation*, like crackling snow, which, as was shown at operation, is not always due to inflammation of the pleura or peritoneum, but at times depends upon an œdema of the liver-cells. Boinet was unable to confirm the latter observation, nor does he believe that the crepitation, when it occurs, is caused by œdema of the liver-cells.

A palpable and audible *friction* was long ago (in 1838) described by Malcolmson,[4] and later by Bertrand[5] and others. It is probably due to perihepatitis or to inflammation of the pleura transmitted through the diaphragm. As perihepatitis may accompany various lesions of the liver and of the general peritoneum, the friction produced by it is not pathognomonic. It will, however, serve to focus the attention upon the hepatic region, and in that way may prove of much value in diagnosis.

10. In a number of instances abscess of the liver has been associated with *right-sided pleural effusion*, the result of inflammation

[1] Lancereaux: Traité des maladies du foie et du pancréas, p. 250, 1899.
[2] Boinet: Revue de Médicine, vol. xvii, p. 57, 1897.
[3] Hassler and Boisson: Revue de Médicine, 1896.
[4] Malcomson: Medico-Chirurg. Trans., vol. xxi, p. 91, 1838.
[5] Bertrand: Gazette hebdomadaire, p. 470, 1890.

transmitted through the diaphragm. Such an effusion must be regarded as a sign of distinct diagnostic value, and its presence in an obscure case of tender enlargement of the right hypochondrium is strongly suggestive of hepatic abscess, or at least of abscess below the diaphragm.

11. The other symptoms, such as *digestive disturbances*—vomiting and diarrhœa—*scanty urine*, *hypazoturia*, *respiratory disorders*, etc.—are often present, but their diagnostic value is merely tributary.

The leucocyte-count, generally of much value in determining the existence or non-existence of suppuration, seems to be of but little help in abscess of the liver. There is no record upon this point in the present case, but in two others that have come under my observation leucocytosis was absent.

That a history of dysentery is of great importance in the diagnosis of hepatic abscess need hardly be further emphasized.

12. The most important agent in diagnosis is, beyond question, *exploratory puncture*, which may be undertaken without fear over the point of greatest prominence; or if there be no special region discernible, in the seventh interspace in the midaxillary line. In any event, the point of election will be governed by the circumstances of the case. The needle should be long and should be plunged in deeply. An aspirator needle or Pravaz syringe may be used. No harm results from making several punctures in different places, if the first fails to reveal pus. In rare instances it has happened that the needle did not penetrate into the abscess cavity, the abscess being discovered only at autopsy.

The nature of the pus, as a rule, at once determines its hepatic source; but there are occasions in which it is difficult to decide whether the pus comes from the liver or from the pleura. In such a contingency the manner in which the pus escapes is of diagnostic value. If the collection is above the diaphragm the flow will be more forceful during expiration, while if it is below it, inspiration through the depression of the diaphragm causes the flow to increase. The same factors influence the trocar, which will be motionless if in the pleural cavity, but will move actively up and down with respiration if in the liver. But while these signs distinguish between supradiaphragmatic and subdiaphragmatic collections, they do not aid us in differentiating between hepatic abscess and a

collection of pus between the diaphragm and the liver (pyothorax subphrenicus).

Treatment.—The treatment of abscess of the liver is operative. Regarding the merits of the various methods of operation proposed, I am not competent to express judgment, and would refer the reader to the admirable work of Waring.[1] I may perhaps be allowed to add, however, that the operation in which the abscess and the pleura are opened at one sitting, so to speak, as was done by Dr. Frazier in the present case, seems to answer well all the requirements, and is, as a rule, preferable to the operation *à deux temps*.

[1] Waring: Disease of the Liver, Gall-Bladder, and Biliary System, 1897.

A CASE OF DOUBLE PAROTITIS, COMPLICATING SUPPURATIVE PYELITIS AND CHRONIC COLITIS. AUTOPSY.

SERVICE OF DR. DAVID RIESMAN.

BY W. C. KLUTTZ, M.D., RESIDENT PHYSICIAN.

There is very little to be found on parotitis complicating other diseases in recent medical literature. Paget's[1] series of articles in 1886 seems to have well-nigh exhausted this subject, and his explanation that a sympathetic relation exists between the parotid gland and the abdominal viscera, or viscera in relation with the peritoneum, has held to the present day.

Another explanation is that given by Tayler[2] in the London *Lancet*, 1886. He thinks that septic parotitis is part of a general sepsis, in as much as it is almost invariably associated with a microbic process elsewhere, and, as a rule, goes on to suppuration. But in nearly all the cases reported and confirmed by post-mortem examination there was only the parotid abscess as the evidence of a general sepsis, without complications in the lung, liver, or spleen, as would be expected if the parotid disease was a part of a generalized sepsis.

It is probable that the bacteria infect the gland through Stenson's duct, the condition of the patient being a predisposing factor. In the majority of instances in which the septic parotitis was observed the patient was in an extremely low state, and as the mouth is normally the habitat of pyogenic bacteria, and in such depraved states probably teems with them, it is not surprising that contiguous glands and cavities (middle ear) may become involved.

Septic parotitis occurs more frequently as a complication of typhoid fever than of any other single disease.

According to Osler,[3] it develops usually in the third or fourth week, and in the majority of cases goes on to suppuration. It does not necessarily imply an unfavorable prognosis.

[1] Paget: "Inflammation of Parotid Gland." London Lancet, vol. i, p. 732, 1886.
[2] Tayler: London Lancet, vol. i, 1886.
[3] Osler: "Parotitis in Pneumonia." University Medical Magazine, 1891.

Pneumonia, gastric ulcer, ovariotomy, dysentery, and traumatic injury to the abdominal wall, are other conditions in which, or after which, parotitis has been observed.

Among rarer affections in connection with which parotitis may occur are mentioned the following: Incision of the cervix (Goodell);[1] passage of instruments into the urethra, accompanied by urethral fever (Cribb and O'Connor);[2] symptomatic of early pregnancy (Hawkins);[3] chronic interstitial nephritis (Legg);[4] compound fracture of the ilium (Pepper);[5] cholecystitis (Atkinson),[6] and surgical kidney.

In the case reported by Atkinson in which the parotitis was a complication of cholecystitis there was a history of a recent attack of typhoid fever.

Osler[7] reports a case admitted to the Philadelphia Hospital in October, 1888, as the only one he had seen up to that time as a sequel to pneumonia. In his case there was suppuration in the parotid gland, as determined by autopsy.

It has been shown by several observers that the secreting glands of the body are actively concerned in the elimination of micro-organisms, and that the salivary glands, as well as the liver and kidney, participate in this function. If this is true, it is not unreasonable to assume that some of the cases of parotitis are due, not so much to bacteria entering by way of Stenson's duct or to a general septic infection, but to an effort on the part of the gland to eliminate bacteria that have entered the blood from some primary septic focus.

If this theory is correct we would, *a priori*, expect to have at times a septic pancreatitis.

We have no data at hand showing that this occurs, but a careful search for the condition might at some time be crowned with success.

The history of the case we have to report is as follows:

Edward Riley, aged seventy-two years, an American by birth and a laborer by occupation, was admitted to the hospital from the Out-wards on July 16, 1900, his chief complaint being incontinence of urine. He had had this for some time, and was compelled to rise at night to pass water. On account of his mental state—he

[1] Goodell: "Inflammation of the Parotid Glands following Operations on the Female Genitals." Transactions of the American Gynecological Society, New York, 1885-'86.

[2] Cribb and O'Connor: London Lancet, 1886.

[3] Hawkins: London Lancet, 1886.

[4] Legg: Transactions of Pathological Society, vol. xx. London, 1868-'69.

[5] Pepper, A. J.: British Medical Journal, vol. i. London, 1878.

[6] Atkinson: Johns Hopkins Hospital Bulletin, vol. viii. Baltimore, 1897.

[7] Osler: Loc. cit.

was semi-demented—it was impossible to obtain a more definite history. He had also suffered from diarrhœa in the Out-wards, which persisted for two days after his transfer to the hospital.

Status Præsens.—He is emaciated and poorly nourished, with prominent malar bones. There is complete corneal leucoma of the left eye, and an arcus senilis in the right eye. The temporal arteries are very tortuous, and the radial arteries are distinctly calcareous.

The tongue is black over a small area on the dorsum. The patient has a peculiar habit of spasmodically contracting the superficial muscles of the neck, comprising the thyro- and genio-hyoids, causing transverse wrinkling of the skin. It is probable that the platysma is also involved in this contraction. Associated with the spasm is a persistent sniffling. The supra- and infra-clavicular fossæ are quite marked.

Heart.—The apex-beat is neither seen nor felt, but there is a well-marked pulsation in the epigastric region. On percussion the outline of the heart is not enlarged.

At the aortic cartilage there is a systolic murmur which is low-pitched in character and not transmitted into the vessels of the neck, or elsewhere.

The sounds at the apex are very feeble, and with the first sound there is a murmur which is heard with maximum intensity at a point half an inch within the nipple line. It is harsher than the murmur heard at the aortic cartilage, and is not transmitted to any distance.

Lungs.—There is only a moderate degree of respiratory excursion, which is the same on both sides. Vocal fremitus is increased. The percussion-note is hyperresonant, and of a peculiar band-box quality. Over the entire abdomen, including the prominence in the hypogastric region, there is a tympanitic note. The lateral veins are rather prominent and the skin shows general œdema. The epigastric pulsation is situated a little to the left of the ensiform cartilage, and at this point there is heard on auscultation a heavy, booming sound. Palpation of this area does not give an expansile thrill. The pulsation is not found in the back or elsewhere. The hepatic dullness extends from the fifth interspace above to the costal margin below.

The spleen as determined by percussion extends from the eighth interspace posteriorly forward to the anterior axillary line.

The plantar reflex is active, and irritation of the sole of the foot causes normal flexion of the toes.

There is some prominence of the veins all over the legs.

The patient was catheterized, and six ounces of residual urine were withdrawn.

A rectal examination shows moderate enlargement of the prostate, and gives a great deal of pain, but is otherwise negative.

The patient imagines himself in the House of Correction.

Two days after admission the diarrhœa ceased.

The patient gradually became unconscious, and on July 20, 1900, a decided swelling of the left parotid gland, very tender to the touch, developed. It apparently did not fluctuate, but the determination of this was interfered with by the overlying œdema.

Exploratory puncture in several parts of the gland yielded only a few leucocytes. There was beginning swelling of the right parotid gland just below the ear.

July 21, 1900.—The patient is much weaker, and the pulse hardly perceptible. The surface of the body is covered with cold, clammy sweat, and there is cyanosis of the arms.

A microscopic examination of the fæces showed fat and vegetable cells, some muscle fibre and sulphide of bismuth crystals.

The urine on admission had a specific gravity of 1003, was yellowish in color, acid in reaction, and an examination of the sediment microscopically showed it to be largely composed of pus.

The chemical examination, using the contact test, gave a distinct ring of albumin; sugar was not present.

The specimen obtained by catheterization was amber in color, and turbid, acid in reaction, and deposited a white, flocculent sediment. There were large epithelial cells, probably of bladder origin, on examining the sediment microscopically, and a large amount of pure pus as well as micrococci. The contact test showed a decided ring of albumin.

A blood-count was made on the 19th, the fourth day after admission, and gave 3,200,000 red blood-cells and 14,000 leucocytes. The hemoglobin was not estimated. The parotid glands continued to swell, and were extremely tender, so that even while the patient could not be roused to speak, the slightest pressure on the glands caused him to wince. He died on July 21, 1900. The clinical diagnosis made by Dr. Riesman was mitral regurgitation, arterio sclerosis, suppurative pyelitis, and parotitis.

The autopsy was performed by Dr. J. D. Steele. The notes are briefly as follows:

There is marked swelling of the parotid gland on both sides, more marked on the left side.

The relations of the abdominal cavity were normal in the general examination.

The thoracic cavity showed nothing of significance in the lungs and pleuræ.

The heart was about the normal size.

The left ventricular muscle was 14 mm. in thickness, and the muscle was pale and rather friable, suggesting cloudy swelling. No interstitial change was apparent.

The mitral valves showed several atheromatous plates along the base of the anterior leaflet, and the chordæ were shortened and somewhat thickened. There were also some opaque and yellow plates in the aorta above the valves.

The valves were all competent. The valves of the right side were normal.

The spleen was somewhat larger than normal and the pulp was dark and soft.

The stomach was in its normal position and of usual size. Its mucous membrane was thickened and rough near the pylorus, but the cardia was so affected by post-mortem digestion that its condition could not be recognized. The small intestines were normal. The colon showed numerous ulcerations and recently-healed scars affecting particularly the sigmoid and descending colon.

The ulcers varied in size from 2 cm. in diameter to pin-point erosions of the mucous membrane. The lymphatic glands lying in the meso-colon were decidedly enlarged and many were as large as a cherry.

The adrenals were normal.

When the posterior layer of the peritoneum was cut through, the ureter upon the right side was found to be somewhat dilated from the pelvis of the kidney to the bladder. The dilatation was greatest nearest the pelvis of the kidney, where it was about 3 cm. in diameter. It gradually diminished in size until the ureter entered the bladder, where it was about twice the size of a lead-pencil.

The ureter upon the left side was slightly dilated along its whole course to about the size of a lead-pencil.

The bladder was small with thickened walls. The transverse bands upon the bladder wall were very marked, and the mucous membrane was thick and rough.

The bladder contained four ounces of purulent and offensive urine. The ureters also contained pus.

The prostate was enlarged, especially the middle lobe, and seemed large enough to cause considerable obstruction to the emptying of the bladder.

The right kidney was swollen and large. On stripping of the capsule the cortex was mottled and yellow with small whitish areas, which on section seem to be small collections of pus.

The capsule was somewhat adherent.

The section of the cortex was found to be thin and pale, with many yellow areas and with numerous small abscesses.

The pelvis was dilated and contained much pus, and the calices were also dilated, and extended far into the secreting substance of the organ.

The left kidney was in a similar condition but not to the same extent. There were numerous small abscesses and dilatation of the calices.

The gall-ducts were free.

The liver was not enlarged. It was pale and friable and suggested cloudy swelling. The pancreas was normal.

Next to the parotitis, the most interesting point in our case was the suppurative pyelitis.

The general condition of the patient, the fever, and an acid urine with pus, suggested pyelitis, but not until the development of the parotitis did we feel sure of the diagnosis. The diarrhœa was of such short duration that it was not considered to bear any relation to the parotitis.

The occurrence of secondary parotitis is of decided value as a diagnostic point, in that it nearly always suggests some abdominal condition. In obscure cases of continued fever it would suggest typhoid fever, inasmuch as typhoid fever is the commonest condition causing it.

Janowski[1] has made mention of a case of typhoid fever with secondary parotitis as a complication, in which the typhoid bacillus alone was found as the cause of the abscess formation.

Another interesting factor in our case was the habit-spasm. In discussing this spasm with Dr. Riesman, he mentioned a case from his practice in which the three movements of head-nodding, blepharospasm, and sniffling were associated, although occasionally the latter two occurred without the head-nodding.

A few years ago the black tongue was a much-discussed subject. A scraping was made from the black area on the dorsum of the tongue in this case, and when examined under the microscope showed degenerated epithelial cells and a few bacteria, but no fungi.

[1] Janowski: Centralblatt f. Bakt. u. Parasitenkunde, vol. xvii, No. 22, p. 785, 1895.

A CASE OF CEREBELLAR TUMOR.

SERVICE OF JAMES HENDRIE LLOYD, M.D.

Reported by T. PERCEVAL GERSON, M.D., INTERNE.

F. E., white; male; aged twenty-one years; native of Philadelphia; for a time occupation that of a sailor. Admitted to the nervous department of the Philadelphia Hospital February 23, 1898, in a semi-stuporous condition.

Family History.—Negative.

Personal History.—At his birth labor was prolonged and very difficult, the head being subjected to considerable pressure although no instruments were used. In childhood he had measles and whooping-cough, and at ten months, probably from an infection thought to have been acquired at the breast of a wet-nurse, he developed upon the skin a scattered papulo-pustular eruption. He was never afterward seriously ill until the appearance of his present malady. In 1893, at the age of sixteen years, he was admitted as a student to the Pennsylvania nautical school-ship "Saratoga," having passed, it is said, a creditable entrance examination. He made but one cruise, lasting seven months, and was discharged, it is thought, for stupidity and insubordination. The tobacco and alcohol habit, which he contracted while aboard ship, was continued afterward in excess. He contracted gonorrhœa but there is no history of syphilis. It was noticed by his family and friends that he became unexplainably irritable, reticent and melancholy, complained frequently of headache, had very little energy and would sleep for hours at a time, these symptoms probably all pointing to the incipient stage of the pathological growth found later. There was never obtainable a history of traumatism.

History of Present Illness.—It was in August, 1897, that it was first noticed that the patient suffered from intense headache; instead of his customary brightness he became dull mentally, would lean over for hours holding his forehend and occiput in his hands; became extremely melanoholy, and in the following December his eyes began to assume an expressionless stare, sight gradually failed, hearing became impaired and later also failed. Taste and smell were never known to have been affected. Appetite until two weeks preceding admission had been good, bowels regular, and after admission he developed incontinence of urine and fæces. For some time preceding admission it had been noticed that he was disinterested practically entirely in his surroundings, would walk along with bowed head, globes of eyes directed upward, his movements being uncertain and frequently leading him to stumble into objects. Early in the course of his disease he seemed unwilling to converse, because of a sluggish mentality; later it was seen that this was due undoubtedly to his absolute loss of hearing. At no time did he have true delusions, hallucinations or convulsive seizures, or any palsies. After January, 1898, he developed an even more profound condition of stupidity. He, however, would occasionally blurt out his wants. Shortly before admission to Blockley he received treatment at the Pennsylvania Hospital. A note from that hospital gives the result of the eye examination which is appended below.

Physical Examination.—Patient shows considerable emaciation. Decubitus on right side with knees flexed and thighs drawn up on abdomen, chin resting on chest and hands clasped on occiput and forehead, seemingly because of pain. The patient usually is quiet, sleeping a great deal. The skin is dry and harsh. The eyelids move involuntarily, but the patient does not wince when an object is rapidly passed to and fro in front of the eyes. The tongue is coated over the mid-dorsum with a heavy yellow fur; breath foul. The patient is utterly oblivious of sound, even when a metal vessel is forcibly beaten in close proximity to either ear. Occasionally he cries out in a plaintive manner his wants, his sentences invariably showing cerebral incoördination. When touched he asks what is wanted or makes the statement that he is awake, etc. Many times during the day or night he makes complaint of the pain in his head. No scars or other signs of trauma on the head were ever discoverable. The patient can scarcely stand. Even when he is supported on either side there is marked tendency to pitching forward. He takes preferably liquid food, but has not perfect control over deglutition, choking invariably on either liquids or soft foods. He has had to be watched, as on a number of occasions he fell from bed.

Examination of Eyes made by Dr. Harlan.—"O. D. *V.* 15/40; pupil responsive; disc swollen 4 D. Venis much engorged, remains of old hemorrhage. O. S. 15/70, same as O. D., except fresh hemorrhage above disc, subjective examination impossible on account of violent headache. Only history obtainable is headache, now improved, vision and hearing having been impaired for two months."—Pennsylvania Hospital (case diagnosed as intracranial tumor).

Eye Examination, February 24, 1898, by Dr. Charles A. Oliver.—Diagnosis, cerebellar tumor. Pupils are dilated ad maximum. Irides irresponsive to light. Pupils vary in accordance with movements of the eyeballs. Marked choked discs, dense. Retinal arteries reduced to threads, and corresponding veins extremely small, the swelling not being very high, but the nerve-tissue being compact. No fixation of eyes upon any object moved into ordinary visual field-blind.

Ear Examination, March 7, 1898, by Dr. George Morley Marshall.—"Membranes were found intact, freely movable. There is no inflammation. No evidence of aural complication."

Chest is spare, expansion symmetrical. The boney landmarks and the surface hollowings consequent upon emaciation are prominent. Costal angle about 80°.

Lungs are normal.

Liver-dullness normal in extent.

Splenic-dullness not distinguishable.

Heart: apex-beat neither visible nor palpable; no murmurs; action slow and regular.

Abdomen examination, negative. Numerous small white pits or scars were observed, the same as on chest and extremities.

Prepuce of penis shows a few small white scars.

Reflexes: Patellar-jerks have the peculiarity of changing in intensity, being either normal, or minus, or plus, or differing on the two sides, at different times. Patellar-clonus and toe-jerk absent. Ankle-clonus very faintly present on left side, but readily elicited on the right. The patient moves body and limbs about apparently at will. Plantar reflexes sluggish. Sensation to the prick of a needle is considerably delayed in the extremities, before response sometimes as many as five seconds elapsing. It is somewhat more acute on the neck and face. While in

Cerebellar tumor. (Sarcoma.)

the wards the patient had two or three spells of vomiting, one of which was distinctly projectile in character, the contents of the stomach being forcibly emitted to some distance from the patient. A comprehensive study of the pain and temperature sense were, of course, impracticable because of the man's mental state. The treatment consisted of thirty grains of potassium iodide during the day, bismuth and salol for the diarrhœa which appeared, and a diet composed of milk, eggs, toast and broths.

The headache never seemed to increase in severity; if anything it decreased, as the patient complained less than upon admission. He became gradually weaker and more anæmic, and there was difficulty in feeding him owing to his choking. Toward the end the respirations assumed the Cheyne-Stokes type, the pulse became very rapid (156), temperature from the normal or subnormal range, where it had mostly been since admission, rose to $102\frac{2}{5}°$, $103\frac{4}{5}°$, etc. The patient died 5 P.M. on April 3, 1898.

Necropsy made by Dr. Lloyd and Dr. Bowman.—Body of a tall male, much emaciated, having several bedsores over the sacrum. There is tattooing upon the flexor surface of the left forearm. On opening the abdominal cavity the walls were noted to be thin, muscular, of dark color. Intestines contracted. Usual amount of peritoneal fluid present. The appendix was very long, the distal end being curled upon itself. The sigmoid flexure was long and distended with gas.

The thoracic cavity: Pleuræ showed no adhesions.

Diaphragm extended to fourth interspace on both sides.

Pericardium contained about four fluidounces of a yellowish fluid and was normal.

Heart, 250 grammes. Right auricle distended with clots, extending into the great vessels. Mitral orifice admitted three fingers; valves, normal. Right ventricle distended with clotted blood. Left auricle contains some clotted blood. Left ventricle was empty. Tricuspid orifice admitted two fingers, valves slightly thickened, showing areas of reddening. Along the free margin of the valves there was marked reddening in distinction to rest of valves, no vegetations. Arch of aorta was apparently normal. Heart muscle was pale and flabby; wall of left ventricle one-half inch thick.

Lungs, left, 510 grammes; crepitant, the lower portion of lower lobe congested, slightly œdematous. Right, 780 grammes. External surface of lower lobe presented an area two inches in diameter, dark-red in color; at its centre is a depressed suppurating area one-half inch deep. The lung crepitates throughout. Beneath the suppurating area referred to were other smaller similar areas.

Spleen, normal, weight 110 grammes.

Suprarenal capsules, normal.

Left kidney, 170 grammes, large; capsule stripped fairly well when cut; dark-red in color, cortex slightly narrowed. There was considerable fat in the pelvis. Right kidney, 140 grammes. External surface bore a small white nodule, calcareous; kidney is smaller than its fellow, otherwise similar.

Ureters and bladder, normal.

Liver, 1570 grammes, slightly enlarged, cut with slight resistance, firm, otherwise normal.

Gall-bladder was normal.

Brain, weight 1390 grammes. A very large tumor was found lying upon the vermiform process of the cerebellum, immediately beneath the tentorium. By

careful dissection this tumor was easily shelled out of its bed. It was found to have only the slightest connection with the brain-substance. In other words, it was encapsulated and had sprung entirely from the membranes. By pressure downward it had flattened and entirely destroyed the appearance of the quadrigeminal bodies and the vermis of the cerebellum, but the aqueduct of Sylvius was pervious, and the anterior medullary velum was unbroken. The tumor was nodular, especially its anterior superior aspect, where one large nodule, the size of a grape, had made pressure into the left cerebral hemisphere. The tumor was very vascular, and was six centimetres wide in its greatest transverse diameter, six and one-half centimetres long in its antero-posterior diameter, and four and one-half centimetres in its vertical diameter. On section it presented the appearance of a sarcoma. Its most striking characteristic, next to its size, was its comparative freedom from connections with the surrounding brain-substance. There was no infiltration of sarcomatous tissue in the neighborhood. The ventricles of the brain were much distended. There were very large Pacchionian bodies along the course of the longitudinal fissure, deeply imbedded in the vault of the cranium.

CULTIVATION OF THE ASPERGILLUS ON URINE.

By L. NAPOLEON BOSTON, M.D.

The frequency with which one detects the presence of the penicillium glaucum, yeast, and mycelium in the urine voided by inmates of this institution prompted me to investigate the relation existing between these and other fungi, which are in many respects similar. For this purpose the aspergillus fumigatus and the aspergillus nigra were cultivated on urine, as follows: Acid, alkaline, and acid diabetic urines were placed in culture-tubes in quantities of 10 to 15 cc. each. Where the urine was found to contain albumin it was heated sufficiently to precipitate this body, after which it was filtered and then placed in tubes, as above stated. One-half of all the tubes were heated at a temperature of 212° F. for forty-five minutes. Tubes prepared in this manner were inoculated with the aspergillus fumigatus, care being taken to introduce, as nearly as possible, the same quantity of the growth at each inoculation, always cultivating the organism on acid, alkaline, and acid diabetic urines. Two tubes containing urine of each variety, one of each being sterile, were used. The same precautions were observed in the study of the aspergillus nigra. All inoculations were made April 13, 1900, and the following is a record of the changes observed. Cultures examined daily to May 13, 1900.

Series No. 1: Aspergillus Fumigatus.—Sterile acid urine, kept at room temperature, four days after inoculation presented a surface growth, which was not easily broken by shaking the tube. Microscopic study detected clumps of mycelium, showing slight budding. Many spores were found, often arranged in large clusters. There were also groups of mycelial threads arranged in the form of an octopus, and as is shown in Fig. 1. On the sixth day the urine was alkaline, of an amber color, and showed a heavy precipitate.

Acid urine was rendered alkaline in four days (possibly due to bacteria). Growth less pronounced than on sterilized urine. Micro-

scopically, the growth was composed of amorphous material, few mycelial threads, and spores. Urine of an amber color.

Sterile alkaline urine presented a less marked growth on surface than did acid urine, which sinks on shaking the tube. On the sixth day alkalinity had increased, and the faint growth on the surface contained few spores, while the color was slightly deepened. On the twenty-third day the surface and upper fourth of the urine presented a chocolate color, and on the thirtieth day this color had extended to the bottom of the tube. The sediment and surface growth, at this time, contained few spores.

Alkaline urine presented a more marked growth, which was found to be largely composed of spores. Alkalinity did not increase until

FIG. 1.

From cultures of aspergillus fumigatus on acid urine. Queen objective 1/6; eye-piece II.

the tenth day, when a browning at the surface of the liquid was observed, and increased gradually to the sixteenth day, at which time the entire urine was of a blackish-brown color.

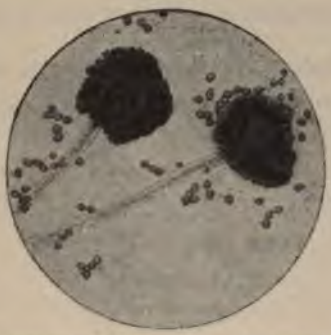
Sterile acid urine (diabetic) presented a whitish growth covering the surface in forty-eight hours, and on the fourth day tipping and shaking the tube did not displace the urine, and the growth was beginning to acquire a greenish hue, while the urine remained an amber color. Microscopic study detected many mycelial threads tipped at one extremity by a sporangium and spores—the usual findings where the aspergillus fumigatus is cultivated on potato, bread-paste, or other media adapted for its vegetation. On the ninth day the growth was thickened, wrinkled, separated from the side of the tube, and its surface studded with sporangia. There was practically no difference in

the action of this fungus when grown on urine that had not been sterilized.

Series No. 2: Aspergillus Nigra.—Sterile acid urine showed marked white growth on surface the fourth day, which prevented the urine from flowing when the tube was tipped. This growth is composed of mycelial threads, many of which present budding. Spores are also present in large numbers, and the octopus formation of the threads is common. Reaction neutral.

Acid urine presented both a growth on the surface and a heavy sediment. Many spores are present in the surface growth, which was largely composed of the mycelium. Reaction alkaline, and the color of the urine was unchanged.

FIG. 2.



Aspergillus nigra from culture on acid (diabetic) urine. Queen objective 1/6; eye-piece II.

Sterile alkaline urine presented but slight surface growth on the fourth day, and by the end of the first week this growth had sunk to the bottom of the tube. Mycelium did not develop, and the spores were of the usual dark color. Reaction neutral after fourth day, and at the surface the liquid was of a cherry-red color.

Alkaline urine differed from the sterilized in that it was rendered highly alkaline, and at the thirtieth day the upper half was changed to a dark brown.

Acid diabetic urine presented the same changes noted in the study of the aspergillus fumigatus, except that the culture medium was changed to a dull black.

A review of the literature on aspergillosis, by Rothwell,[1] credits

[1] Dissertation on Aspergillosis, Victoria University, Manchester, Eng., 1899.

Renon[1] as being the first to call attention to the presence of mycelial threads and spores of this fungus in the urine of animals suffering from experimental aspergillosis (pseudo-tuberculosis). This author found that in from twenty-four to forty-eight hours after inoculation he was able to cultivate the aspergillus fumigatus from the animal's urine, and in most instances he found mycelium—these findings being more constant as the disease progressed. At autopsy these animals were found to present classical lesions of the bladder and kidneys. Renon attributed these lesions to infection through the venous blood supply, as his attempts to cultivate the aspergillus on urine proved that it had little tendency to vegetate when kept at incubating temperature.

FIG. 3.

From urine (diabetic) during an attack of cystitis. Queen objective 1/6; eye-piece II.

Many writers on this subject have called attention to the fact that the aspergillus and other closely allied fungi develop best on acid medium at a low temperature, and the same has proven true in my studies of the organism on urine, which probably explains the wide difference in the findings of Renon and the writer. My review of the literature has been rather hastily accomplished, and possibly some records have escaped my notice. However, I have been unable to find any special record of the effects of this organism on human urine. It has been my privilege to study two cases of cystitis where the urine was loaded with the products of this organism. In one instance (Fig. 3), that of a diabetic, the aspergillus fumigatus was found to be in a most vigorous state of vegetation, while in the other case (Fig. 4),

[1] Comptes rendes des séances et memorires de la Société de Biologie, Feb. 9, 1895.

which presented more acute symptoms—acid urine free from sugar and albumin—the organism appeared to grow with less vigor. Both cases

FIG. 4.

From urine during an attack of cystitis. Queen objective 1/6; eye-piece II.

yielded to treatment, and both local and general symptoms subsided with the disappearance of the mycelium from the urine.

LOBAR PNEUMONIA COMPLICATED BY CARDIAC ABSCESS DUE TO THE BACILLUS OF FRIEDLANDER.

By L. NAPOLEON BOSTON, M.D.

H. W. S., male, aged thirty-three years, had been a patient in the nervous wards of the hospital for the past two years, after which he was transferred to the medical department by reason of endocarditis. Physical examination by Dr. Packard, ten days before death, disclosed the following: Apex-beat in the sixth interspace at the left anterior axillary line; area of cardiac dullness increased. At apex are heard two blowing murmurs—one occupying the whole of systole, the other the whole of diastole. At aortic area there is a loud systolic murmur which is very poorly transmitted, and there is reduplication of the second sound. There was slight change, if any, in the patient's condition for nine days, when he was noticed to be nauseated and developed several paroxysms of vomiting, which were accompanied by marked prostration. He died eighteen hours later.

Necropsy.—The heart is moderately enlarged. Pericardium, normal. The tricuspid and pulmonic valves present nothing abnormal. The chambers of the right side of the heart contain chicken-fat-clots. The aortic valves show vegetative endocarditis involving especially the anterior and middle leaflets. These vegetations are about 2 mm. in size and extend just below the edge of the leaflets which are fused. At this point of junction there is moderate thickening and calcarious infiltration. On section the cardiac muscle presents a yellowish appearance; yet the muscle substance feels quite firm. At a point in the posterior wall of the left ventricle, about an inch above the apex, there is an area the size of a dime, in which the heart-wall is thin and depressed. On the ventricular aspect this area presents a glistening, fibrous appearance. On section it is found to consist almost exclusively of fibrous tissue, extending to within a line of the pericardium. Tricuspid orifice admits three fingers easily, while mitral admits two fingers with some difficulty. The wall of the right auricle shows thickening which is somewhat triangular in outline, and on section there appears to be some new formation within the auricular wall, covered externally by pericardium, internally by the muscle and endocardium. At the centre of this area is a small amount of pus. There is moderate sclerosis at beginning of the aorta. Coronaries appear normal.

Left lung is firmly adherent, the result of an old pleurisy. In the portion of the organ overlapping the heart there is an area of consolidation 5 x 4 x 2½ cm. The surface of this area is red and covered by a moderate amount of lymph, and on section shows the general characteristics of lobar pneumonia; the base presenting a gray appearance, while the apex is seen to be in a state of red hepatization; at the junction of these portions is seen a small abscess, size of a large pea, filled with greenish pus. Elsewhere the lung is normal.

Right lung is completely consolidated at its base, occupying about one-third of the inferior lobe, over which the pleura is darkened and slightly granular. On section the cut surface presents the characteristics of beginning gray hepatization. Near the centre of this consolidation is a small cavity with irregular walls and a fairly well-formed limiting membrane, which is partly filled with croupous exudate. Other viscera showed no gross lesions.

Bacteriology.—Cover-slip preparations made from pus of heart's abscess, abscess of lung, and from area of red hepatization were alike in showing the presence of bacilli occurring singly or in chains of three or four. Rarely were they seen to be nearly circular in outline. These bacilli stained readily by analine gentian violet, but gave up this stain when treated by Gram's method. Each organism presented a distinct capsule, as shown by the accompanying plate.

Pus from cardiac abscess. Queen objective 1/12; eye-piece II.

Cultures were made on agar-agar from abscess of heart, pneumonic areas, and pulmonary abscess, and were alike in developing a profuse smeary, whitish growth within thirty-six hours, at a temperature of 37° C.; later this growth acquired a slightly brownish-yellow tint. Further study showed this organism to be a pure culture of the bacillus of Friedlander. Cultures made from the cavities of the heart, on Löffler's serum, agar-agar, and nutrient bouillon, gave negative results, leaving room to question the cause of the ulcerative endocarditis; however, it cannot be regarded as a case of direct extension from the lung, by contiguity of tissue, since the parietal pericardium was found normal. A similar case[1] occurred in the hospital, where autopsy on an adult male, dead of cerebrospinal meningitis, revealed the presence of an abscess in the wall of the left auricle, due to the diplococcus intracellularis of Weichelbaum.

[1] Medical Record, Sept. 2, 1899.

THE BACTERIOLOGY OF ERYSIPELAS.

By GEORGE E. PFAHLER, M.D.,
ASSISTANT CHIEF RESIDENT PHYSICIAN.

The investigations forming the basis of this article were made upon ninety-eight cases of erysipelas occurring in the wards of the Philadelphia Hospital, under the care of Dr. Daniel E. Hughes, Chief Resident Physician, to whom I am indebted for the clinical observations and many suggestions.

As early as 1870, cocci were found in the skin and blood of erysipelas by Lukowsky, Tillmanns, Wild, Ostle, and V. Recklinghausen, but more positive results awaited the technique established by Koch.

In 1882, Fehleisen described streptococcus as the specific cause of erysipelas, which was later investigated by Rosenbach, Passet, Garic, Achalum, Lutinski, and others. On account of the likeness of the streptococcus of Fehleisen and the streptococcus pyogenes, Rosenbach questions the specific nature of the former organism. Through the investigations of Hajek, the lesions produced by inoculation with either the streptococcus of erysipelas or the streptococcus pyogenes were found not to be constant. Bonne and Bondini Uffreduzzi have studied two cases[2] in which staphylococci were found; one in which death resulted in a case of erysipelas of the face, the staphylococcus aureus was found in the vesicles of the skin, in the lymph chambers, in the blood and in internal organs in pure culture; and the nature of it was proved through culture and inoculation. In another case of phlegmonous erysipelas the staphylococcus citreus was found, together with a few streptococci, but the latter were not found in culture. As a result of these investigations they came to the conclusion that erysipelas is etiologically not a specific disease. As a rule it is due to the streptococcus pyogenes, but may also be caused by the staphylococcus pyogenes, and that the difference in the effects of the pyogenic

[1] Mikroörganisman bei den Wundinfections krankheiten des Meuschen, 1884.

[2] Beitrag zur Aetiologie des Erysipels, Giorn della R. acad. di med. di Trennio, 1886. Ref. ins. Centralblatt für Chirurgie, 1887, S. 134.

organism is due to the difference in the locality and in the degree of virulence, as well as the number of micro-organisms.

Fehleisen, after describing the streptococcus,[1] states that in the progressing border there are also numerous diplococci present. The question now arises as to whether the streptococci or the diplococci were the cause of the erysipelas.

Without reviewing in detail the further investigations, it suffices to state that the streptococcus has been generally accepted as the cause of erisipelas. Dr. Roswell Park, in the last edition of his text-book, says, "It is now definitely established that the infecting organism is a streptococcus."

In a preliminary report published in the *Philadelphia Medical Journal*, January 13, 1900, I described an organism which I had found in pure culture in eight successive cases of erysipelas, and which I called a "Diplococcus." This term was used more in a descriptive than technical sense. I have now studied ninety-eight cases, with special reference to their etiology. The diagnosis in each case was confirmed by Dr. D. E. Hughes, Chief Resident Physician.

The result of my investigations may be stated briefly as follows:

1.	Number of cases studied	98
2.	Growth of bacteria obtained upon the artificial medium	88
3.	Number in which a second inoculation was necessary to obtain growth	5
4.	Number of cases in which diplococci were found	86
5.	Diplococci were found in pure culture in	66
6.	Mixed cultures were obtained in	20
7.	In the mixed cultures streptococci were found in	10
8.	" " " staphylococcus aureus in	7
9.	" " " staphylococcus albus in	2
10.	" " " bacilli in	2
11.	The staphylococcus aureus occurred alone in	2

In nearly all of my cases some antiseptic application had been made previous to the bacteriological examination. This probably assisted in the elimination of foreign organisms from the surface of the skin.

The cultures were made as follows: The ointment or antiseptic was removed and the skin cleansed by repeated scrubbings with cotton saturated with ether. The inoculations for artificial culture were made from the vesicle by piercing with a sterile needle, then carrying

[1] Die Aetiologie des Erysipels, Dtsch. Zeitsch. f. Chir., xvi, 1882.

a loopful of the clouded serum into the culture medium. From the inflamed skin, cultures were made by passing a needle through and beneath the skin about one-fourth inch near its progressing border, then passing serum and blood from the tissue, and inoculating by carrying one or more loopfuls of blood into the culture medium. In each case inoculations were made upon blood serum, glycerin-agar, and bouillon.

In a number of cases cover-glass preparations were made from the serum in the vesicles, which showed diplococci in ten cases, streptococci associated with the diplococci in three cases, but never found alone. Streptococci and bacilli were associated with the diplococcus in one case, and diplococci, streptococci, and staphylococci in two.

Cover-glass preparations were made in the majority of cases from the blood exuding from the puncture made in the skin. In twenty-eight cases diplococci, identical in appearance with those found in cultures, were found in the blood specimens, at times in considerable numbers, and again only one or two in a specimen. In some of the cases no organisms were found. Streptococci were found associated with the diplococci in two cases. Case No. 7 ran a somewhat different course from the others. The first inoculation was made on the third day of the disease, which showed a pure culture of the diplococci. On the thirteenth day the scalp was enormously swollen and gave evidence of a collection of pus.

Cover-glass preparations were now made from the blood of the scalp, posterior to the left ear, which showed streptococci and diplococci in large numbers. The organisms occurred both outside and within the leucocytes. Cultures were made as before, which, in twenty-four hours, showed two distinct series of colonies on blood serum. By inoculation from each of these series upon bouillon, obtained from one a pure culture of diplococci, while from the other obtained a pure culture of streptococci; therefore, I believe that the pus in this case was due to a secondary infection by the streptococcus pyogenes.

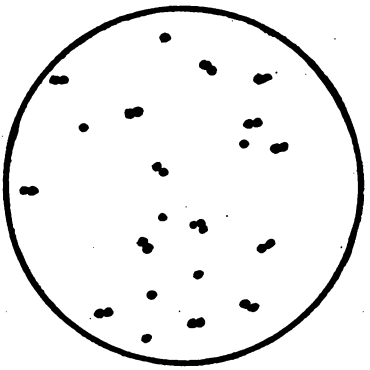
Case No. 4 developed a purulent ophthalmia in connection with the erysipelas. The pus from the eye showed numerous diplococci within the leucocytes, which presented an appearance almost identical with that of gonorrhœal infection, but differing in that the diplococci stain by Gram's method. Cultures were made from this discharge which also showed the diplococcus. A pure bouillon culture was injected

into and beneath the skin of a rabbit; a typical erysipelatous inflammation resulted. The organism was obtained again from the diseased tissue of the rabbit on the third day of the disease. The diplococcus was also found in the diseased skin of this case.

Case No. 45 gave a pure culture of diplococci from the inflamed skin of the forehead. The eyelids gave evidence of pus, and cultures made from this region, as well as blood specimens, showed diplococci and streptococci. Cultures and cover-glass preparations made from the purulent discharge of the eye also showed diplococci and streptococci. Here again the streptococci seemed to be a secondary infection.

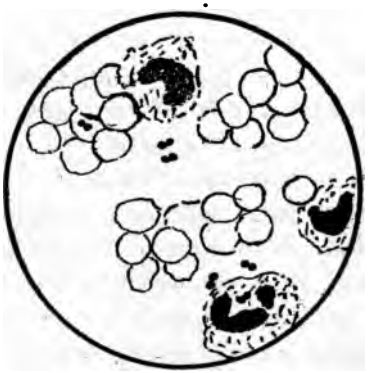
Case No. 3 gave a pure culture of diplococci from the inflamed skin.

FIG. 1.



Bouillon culture as seen with Zeiss microscope, 1-12 oil immersion, No. 4 eye-piece.

FIG. 2.



Specimens of blood from inflamed surface stained with Löffler's athaline solution of methylene blue, 20 m. Zeiss microscope, 1-12 oil immersion, No. 4 eye-piece.

Inoculations made from a large pustule showed three distinct series of colonies, one consisting of diplococci, one of streptococci, and the third of the staple culture. Inoculation made from the healthy skin, about an inch from the inflamed area, with the same technique as before, showed no growth. Inoculations were made from the healthy skin in fifteen cases, but no growth developed. In two cases the staphylococcus albus was found from the healthy skin.

THE DIPLOCOCCI.

Morphology.—Spherical cocci, about the size of the pneumococcus, usually occurring in pairs, often singly, and very rarely a chain of four is seen, which is probably an accidental arrangement of two pairs.

As observed in the contents of the pustules in the blood of the inflamed part and in the blood from the rabbit, it has been seen only in pairs, and at times appears to be encapsulated. It is found both outside and within the leucocytes. This diplococcus is stained readily by aniline gentian-violet, by carbol-fuchsin, and by Grams' method. It does not stain well with Löffler's alkaline solution of methylene blue, and less with a saturated aqueous solution of methylene blue or with Gabbit's solution of methylene blue.

Biologic characters.—It grows in the presence of oxygen and at the room temperature, but better at 31° C. It is non-motile. Cultures upon bouillon show a cloudiness at the end of twelve hours, which increases from day to day, but which tends to clear after several weeks, leaving a sediment.

Glycerin-agar at the end of twenty-four hours shows minute opaque colonies, pin-point in size, whitish in color, and sharply defined in outline after from five to six days; these colonies become about 1 mm. in diameter and slightly raised from the surface. Blood serum shows a more luxuriant growth, the colonies being three to four times the size of those upon glycerin-agar and proportionately raised above the surface. They are of a porcelain-white color, distinctly outlined, and under a low magnifying power appear smooth.

Stick cultures show a beaded growth all along the line of inoculation. Gelatin is not liquefied, and there is no evidence of the production of gas. In stick cultures growth occurs all along the line of inoculation, but apparently cease after from twenty-four to forty-eight hours. There seems to be no radiations and no tendency for the surface growth to spread. A slight capping is seen at the top of the puncture. After forty-eight hours, litmus milk shows no change in reaction after being in the incubator fifteen days. It does not grow upon potato.

Inoculation.—Twenty-four-hour bouillon cultures from twenty-one different cases were injected into and beneath the skin of the ears or legs of rabbits; the injections were usually made into the base of the ear. As a rule, after twenty-four hours, a perfect area of erysipelatous inflammation was seen encircling the entire base and extending one-third the distance towards the tip. After forty-eight hours there was a distinct progression towards the tip and a clear line of demarcation. The third day the line of demarcation was scarcely visible, and there was slight fading of the red mass. At times small vesicles developed.

Cultures and blood specimens were made on the third day of the disease, which showed diplococci identical in morphology with those described in the cultures from the human cases. In only one case did inoculation fail to produce the disease. Inoculations upon guinea-pigs and white rats showed no effect. Owing to the sad death of Dr. Kirkbride, our interested pathologist, the sections made from the diseased tissues of several cases in which death had resulted were not studied and were probably lost. Judging from the foregoing results, it seems that the diplococcus described is the most common cause of erysipelas, or of a disease which, in the light of our present knowledge, cannot be diagnosed from erysipelas. Koch's postulates have been demonstrated with reference to this organism, as follows:

1. It was found in the diseased tissues of eighty-six different cases of erysipelas.

2. It was grown upon artificial media in each case.

3. The disease was produced in twenty-four rabbits by subcutaneous inoculations.

4. The same organism was obtained from the diseased tissues of the inoculated animals. I wish to acknowledge the kind interest and assistance given by Dr. D. E. Hughes, the Chief Resident Physician, and Dr. L. N. Boston, the Bacteriologist to the Philadelphia Hospital.

CLINICAL NOTES ON SOME OF THE HYPNOTICS AND SEDATIVES.

By WILLIAM C. PICKETT, M.D.,
ASSISTANT PHYSICIAN TO THE INSANE DEPARTMENT.

When "no restraint" was the watchword of Pinel's followers, it was easy for the like-sounding phrase of "no sedatives" to become accepted doctrine. The great Esquirol, who said (1838) of restraint that it must be "momentary" only, spoke of calmative medicines in but one paragraph referring to the reported use of opium, and he buried this paragraph in a long dissertation on setons-to-the-neck, surprise-baths, and purgatives. But each of these doctrines was too extreme, and the better sentiment of to-day is reflected in Regis's text-book, which admits the occasional necessity of the camisole, the muffs, and even of anklets, and which, though a small book, has several pages on the hypnotic and sedative drugs.

Opium, the great prototype of these drugs, was employed in mania by Valsalva and Morgagni, and has never wanted adherents through all the period of therapeutic anarchy of which we have spoken. In truth, the modern investigation of hypnotics and sedatives is an effort to secure the opium-calm and the opium-sleep for the excited and sleepless patient in the most direct manner—that is, freed from the well-known physical effects of opium. A pure hypnotic and a pure sedative are the goal of the therapeutist in this direction.

Of the very latest hypnotics, few save methylene blue are used in the Insane Department of the Philadelphia Hospital. Trional, sulphonal, chloralamide, hyoscine—with opium and morphine, chloral and bromide—are our main dependence; and as they are well known and generally used—are standard drugs—it might be thought needless to multiply statistics concerning them; but some of our conclusions are so at variance with the common belief, particularly as to the relative quickness of action of trional and sulphonal, that we publish the following summary of our records, with such corollaries and remarks as seem of most practical use.

These records were begun in January, 1900, when to the most experienced and reliable head attendants of the Insane Department was delegated the task of recording, on special blanks, each dose of trional, sulphonal, etc., prescribed by the physician, the time of admin-

istration, the time when the patient fell asleep, the time when he awoke. In many instances it became a matter of difficult judgment to decide when a patient should be considered "awake" in the sense of being released from the drug's control. We agreed among ourselves that in case the subject was restless and noisy for half an hour or an hour, after having been asleep for an hour or two, he should be pronounced "awake" in our technical sense—that is, we should consider the drug's action at an end, even though further sleep might ensue later. This was in order to learn the *direct* and *constant* sleep-producing effect of the drugs, since, if we took account of subsequent irregular "naps," often continuing into the next day, our statistics would be chaotic.

In brief, we endeaved to answer, above all, these two questions:

1st. "How soon, after taking a certain dose of a particular drug, will my patient fall asleep?"

2d. "How long may I expect this sleep to last?"

	Dose in grains.	Number of doses recorded.	Number that failed to cause sleep.	Average time required to cause sleep. Hrs.	Min.	Average duration of sleep. Hrs.	Min.
Trional	30	877	13	1	55	6	22
	20	34	1	1	40	6	22
	15	10	00	1	54	6	20
	Total,	921	14	1	54	6	22
Sulphonal	30	66	3	1	17	3	3
	25	43	14	1	50	4	3
	20	561	106	1	57	5	19
	15	273	71	1	45	4	37
	Total,	943	194	1	50	4	54
Chloralamide	45	353	33	1	32	3	23
	40	420	182	1	38	3	35
	30	641	171	2	3	4	34
	20	51	25	1	55	4	11
	Total,	1465	411	1	48	3	54
Hyoscine, t. i. d. (Effect of three doses.)	$\frac{1}{50}$	600	00			5	53
	$\frac{1}{100}$	1152	00			6	2
	Total,	1752	00			5	59
Lithium Bromide, t. i. d. (Effect of three doses.)	30	118	22			4	56
	25	113	00			7	50
	20	25	00			8	11
	Total,	256	22			6	28

It would appear from these records that trional is no quicker in its action than sulphonal, and that the sleep from trional lasts longer than that from sulphonal. Both of these conclusions are opposed to the common teaching; but our first contains a fallacy, in that the cases represented in the trional record were the very worst—acute mania and the maniacal states—in which we have learned not to rely upon sulphonal. Doubtless, in any given case, trional will prove more rapid in its action.

Thus we find that trional in 167 cases induced sleep in one hour or less, in 62 of these the period being a half hour or less; whereas, with sulphonal one hour was sufficient in only 143 cases; a half hour in 15 cases. Again, the number of failures from sulphonal is 194; from trional, 14. Expressed in percentage, these facts may be tabulated as follows:

	Sleep resulting in one hour or less.	Sleep resulting in half hour or less.	No sleep resulting.
From trional.....	18%	7 %	1½%
From sulphonal.....	15%	1⅘%	20 %

The lasting effect of sulphonal, so much dwelt upon by authors, pertains, we think, to its *sedative* action, in which it far surpasses trional. An excited patient who took sulphonal last evening may be subdued and drowsy to-day, while a similar patient who was given trional is perhaps as noisy this morning as ever, although refreshed by a better sleep than the sulphonal patient enjoyed.

In considering the duration of sleep, one must make allowance for the character of the cases. A dose of trional which produces six hours' sleep in acute mania, would probably give a much longer sleep in the insomnia that one meets in private practice, and for the latter case the dose should be proportionately smaller. This grading of doses will explain the seeming paradox in our statistics that, of trional for instance, fifteen grains give as much sleep to one patient as thirty grains give to another. It is simply that fifteen grains were estimated to be the measure of resistance to trional in the one patient; thirty grains the measure in the other patient. An important conclusion may be drawn from this observation, to wit:—that when we have found the dose of a certain drug which will "put the patient to sleep," no advantage is gained, in this particular case, by doubling or by trebling the dose. Indeed, at one time we were prone to repeat the

thirty-grain dose of trional in obstinate cases, more than one patient having taken even ninety grains in one evening. But the surplus drug caused intoxication rather than true sedation, and the sleep was unsatisfactory; so that Dr. D. E. Hughes has formulated this rule:

"When in the average adult thirty grains of trional fail to bring on sleep, this drug is unsuited to the case."

This rule applies to the other hypnotics in their respective doses, our standard doses being:

Chloralamide	**gr. xxx.**
Sulphonal	**gr. xx.**
Trional	**gr. xxx.**

When trional fails it will generally be necessary to have recourse to the hypodermic of hyoscine, gr. $\frac{1}{100}$, aided and guarded by morphine, gr. $\frac{1}{4}$.

Intoxication, mentioned above, is liable to occur from any of these drugs. Chloralamide, more soluble and more transient in its action, is least apt to cause it, although several times we have seen marked cerebral vertigo—never dangerous—from this drug. Trional may cause sluggishness, drawling speech, and drowsiness; but sulphonal sometimes induces a state so peculiar, so alarming, and so hard to conquer, that it is perhaps wise to speak of this state in some detail.

An excited patient has been taking sulphonal for several days, and perhaps has not been quieted by it. He continues to be garrulous, but his speech has a slight drawl; he is still restless of body, but his motions are somewhat sluggish. He seems in a state of "exhaustion from mania." But now at any hour we may be notified that this patient is much worse, and at the bedside we find this state of things: the patient lying relaxed and covered with sweat; breathing rapid, pulse weak, *abdomen distended.* From time to time he vomits—the vomit of intestinal obstruction—and when we attempt to relieve the tympanites we find that the patient is obstinately constipated. The urine is diminished in quantity and contains a few hyaline casts.

This is a state of collapse with intestinal paralysis, and is, of course, dangerous. To explain it we must assume a cumulative action of sulphonal, but its suddenness of onset makes the drug seem very treacherous. Such untoward effects of sulphonal are not common, and experience with it enables us to foresee danger in the early hebetude which we have described. It has seemed to us that the

main point in guarding against ill effects is to *keep the bowels open*, both to carry off the excess of sulphonal and to avoid auto-intoxication, which probably are joint causes of the toxæmia from sulphonal. Our care to prevent constipation may account for the fact that in some thousand administrations of sulphonal at Blockley, but once has the red coloration of the urine from hænatoporphyrin been observed. In that case the port-wine coloration of the urine appeared about two weeks before a fatal termination from gastric cancer.

After all, sulphonal is a valuable drug in the hands of one who knows its danger-signals. For the refusal of food, so common in melancholia and katatonia, this drug is almost a specific, as was discovered a few years ago by Dr. D. E. Hughes, and as our experience has proved. Sulphonal as a sedative relieving mental tension excels even morphine.

Hyoscine belongs in a different category, and we have not seen the powerful effects from single doses which Magnan describes. We generally employ hyoscine in the dose of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain, three times a day, by mouth, and mostly for the restlessness and irritability of chronic mania, especially of senile mania. It is to this "agitation," as remarked by Magnan and by Agostini, that hyoscine addresses itself particularly. Magnan says: "It is the specific of the syndrome 'agitation.'"

Finally, we commend trional as most generally useful—the safest, surest, most efficient hypnotic with which we are acquainted, approaching nearest to the ideal of a pure hypnotic. Our great need is of a correspondingly safe, sure, and efficient mental sedative.

HOSPITAL TREATMENT OF PEDICULOSIS.

By J. ABBOTT CANTRELL, M.D.

Requests have been so frequent, both at my clinical and private work, for the best, quickest and easiest method for removing pediculi and their effects, that I feel a few words upon the manner adopted by me in private and public practice will prove interesting to my readers.

Why the removal of these parasites should prove so troublesome I am at a loss to state, because they are visible and not obscured as in a case of scabies or as in the vegetable parasitic affections of the skin. That they are difficult to exterminate, the many questions have proven. That the result is easily and quickly gained will admit of no doubt, can positively be determined after a careful perusal of these lines.

As we are all aware, there are three different, although of the same species, parasites which it will be our fort to attack, but whether it is the pediculus capitus, the pediculus vestimentorum or the pediculus pubis, the result in all cases will be similar. We, of course, admit that the methods adopted will differ slightly according to the point of attack, because in the one instance we have the parasite living in the clothing, from which it passes to the skin to receive nourishment, while in the other two it lives upon the hairs, only attacking the skin in its hunger. In the first instance the nits or ova are deposited in the seams of the wearing apparel, while in the latter two the eggs are found attached to the hairs.

The diagnosis is not a difficult problem if one takes the time to carefully observe the hairs in the affected region where the parasites can be seen coursing along each strand, when numerous, but if not plentiful, simply parting the hair reveals their presence. If the parasites are not detectable, we may notice upon the hairs the ova or nits, which are grayish-white bodies with the apex directed toward the proximal end, while the bulbous portions present its base pointed toward the distal. It may not prove so easy to distinguish the presence of the pediculus in the body region, for the very reason that,

unless their attack is suspected, the skin lesions may be overlooked and, consequently, the seams may escape examination. The especial lesion observed upon the skin is a punctate hemorrhagic spot which is about the size of a pin-point, but soon reaches a size which is easily detectable after the incessant scratching indulged by the affected person. In the treatment of this condition I have long since discarded the use of the mercurial ointment, because it is not only not effectual, but I have apparently observed the parasite to grow fat upon it. It does not prove destructive in all instances to the parasite and never, in my experience, to the egg.

The method which I have for some time been using, and the one in which I have received the better and more quick results is of the most simple means, and as it is just as effectual, if not more so, than most of the so-called parasiticides and is far less harmful, I have almost discarded all other remedies.

Taking for granted the appearance of some one suffering from these pests, we have to adopt methods which appear adaptable to the diseased region, but first of all we must dispense with all covering to the part. The person is then given a bath in hot water into which we have previously placed some bicarbonate of sodium, or carbonate of ammonium, one to three ounces to a bath of thirty gallons. These ingredients do not have a destructive effect upon the parasite, but remove the ova from portions of the body or hair to which they may be attached. It is not necessary to cut the hair at all, because this washing will be sufficient to remove the ova, and if carefully performed will remove most of the parasites. The clothing must be placed in different places to kill the parasites and remove the ova. The underwear is simply boiled for a short time, when all evidences are removed. The outer clothing is covered with brown paper and placed in a heated oven for several hours, and this suffices to destroy ova and kill the parasites.

Usually following this, little if any treatment is necessary except to restore the skin to its normal functions, but as the fear may be entertained that all the parasites have not been killed, I may mention the treatment which has always acted in good stead.

The drug which has always proven satisfactory in my hands is the fluid extract of staphisagria. As it is not poisonous to any marked degree, it may be given without fear of consequences. When the

condition affects the scalp it is more preferable to advise it in liquid form, and the preparation used in my work is two drachms of the staphisagra to six ounces of dilute acetic acid. The acetic acid removes the ova or nits. When the condition is observed upon the non-hairy portions of the body, two drachms of the staphisagria is added to one ounce of lard or petrolatum. If any parasites or ova have been left by the washing process these preparations will entirely remove them.

These measures are all that are required to remove the ova or parasites, but the consequences of their presence still remaining we must direct our efforts to cure the complication.

It must be remembered that the parasites do not bite, but that they insert their proboscis, through which they extract blood, their nourishment, and that upon the withdrawal of this instrument the blood wells up into the puncture, after which the itching is developed, and to get relief the affected person scratches and tears at the point of attack until numerous excoriations are produced, which are observed upon the skin as small punctate hemorrhagic spots and as small ulcerations upon the scalp, which soon crusts and matting the hair gives soon a disagreeable odor.

Those observed upon the body need only the treatment which is called for in ordinary dermatitis, such as the ointment of zinc oxide with possibly the addition of a slight amount of carbolic acid to cure the itching. The condition upon the scalp demands more energetic measures. First of all, it is necessary to soak the scalp in hot sweet oil or to poultice with ordinary starch to remove the crusts that are present, and then to use some medicament that will heal the ulcerations. The drug used mostly in my service is the ammoniated mercury ointment, either in full or diluted strengths.

By a careful observance of the measures referred to, there will be no trouble at all in curing every case that may present itself, and I hope that no one will believe it necessary further to shave the part, a measure which I have learned is often adopted, and usually without result, because the habitat of the animal is never treated.

THE BABINSKI REFLEX.

By CLARENCE VAN EPPS, M.D., Interne.

I owe the opportunity to make the following study to my chiefs at the Philadelphia Hospital, Drs. Mills, Dercum, Lloyd and Burr, and to the Chief Resident Physician, Dr. Daniel E. Hughes. The purpose of my investigation was to determine the conditions in which the Babinski reflex is present. The data consisted of one thousand persons, classified as follows:

Babies and children	100
Patients in medical wards presenting no nervous symptoms	165
Insane patients presenting no organic cerebrospinal disease	335
Patients suffering from nervous disease but presenting no symptoms of involvement of the lateral tracts	213
Hemiplegics and diplegics	125
Patients having disease of the spinal cord with manifest involvements of the lateral tracts	62

In health on stroking the sole there is flexion of the toes with or without movement of the ankle and leg. This reflex is not present in every one, some normal persons having no plantar reflex at all.

Babinski discovered that in certain diseases the plantar reflex is altered, and claimed that this alteration is constantly present whenever the lateral tracts are diseased. He described the alteration as follows: There is extension of the great toes with or without extension and separation of the other toes, this movement being slower than a normal reflex and more readily produced by stroking the outer than the inner side of the sole.

In testing for the reflex I have used, as a rule, an ordinary blunt tooth-pick; where the sole was very sensitive I used my finger-tips, and where very strong plantar irritation was necessary I have used the blunt point of a small metal bar. Usually extension when present was better produced by stroking the outer side of the sole in a forward direction. Less often, transverse stroking of the sole was more efficient. When flexion was present, stroking the inner side of the sole usually produced the better reflex.

I have found that there is a great variation in the number of toes which extend, and that sometimes there is flexion of some of the smaller toes and extension of the others, including always the big toe, which is never flexed. For example, in sixty-eight hemiplegics the following manifestations of the Babinski reflex were found:

Extension of all the toes, big toe most	50	per cent.
Extension of the big toe, small toes flexed	27	"
Extension of big toe, no motion small toes	22	"
Extension of small toes only	1	"
Extension of all the toes, small toes most	0	"

To determine the normal plantar reflex I examined five hundred patients from the general hospital and the insane department. None of these patients had any symptoms of organic cerebrospinal disease. In this group I have found the plantar reflex absent on both sides in nineteen cases, or only 3.8 per cent., and absent on one side only in eleven cases, or 2.2 per cent. These figures are much smaller than those of most investigators, many reporting an absence of motion in both feet in 10 per cent., and in one foot only in 15 per cent. On the other hand, Collier[1] states, "It is doubtful whether the plantar reflex is ever constantly and completely absent in healthy subjects." This may be explained by the fact that he has considered as a part of the normal plantar reflex contraction of the thigh muscles, especially the tensor-vaginæ-femoris. Considered thus, I found complete and constant absence of all reflex motion in only one case, or one-fifth of 1 per cent.

In one case there was constant extension of all the toes in one foot; in two cases irregular flexion and extension occurred in one foot only and in three cases in both feet. In one case constant and typical extension occurred in all the toes of both feet; or in all extension in some form was present in seven cases, or 1.4 per cent., and in only one case was a typical Babinski reflex found in both feet. The history of this case I now give in detail:

G. E., male; German; laborer; married; entered hospital September 6, 1900, complaining of cough and abdominal pain. No history of syphilis; typical phthisis history for three years past; tubercle bacilli were found, and patient was sent to phthisis ward, where malarial paroxysms developed.

Examination showed a fairly well-nourished, muscular German of about forty-five years; memory and speech fair. Eyes: pupils equal 2.5 mm., reacted well, to

[1] Brain, Spring, 1899.

light accommodation and convergence, no extra-ocular defect, vision of each eye normal, ophthalmoscopic examination negative. Tongue straight, dry, gray-coated, no tremor; pulse normal, heart showed no gross lesion; lungs showed cavity at right apex and involvement of left apex; abdomen slightly tense and tender; spine straight, no prominences, no tenderness. Extremities were muscular and gross strength normal, no tremors or ataxia, station and gait good, sensation normal. Reflexes: no jaw-jerk, biceps-jerk and knee-jerk equal and quick, no ankle- or patellar-clonus. Stroking the right sole caused active extension of all the toes, big toe most, while stroking the left sole caused active extension of the big toe with indefinite extension and flexion of the smaller toes. With the exception of two tests made during the height of the second febrile paroxysm in which the toes were usually flexed, the patient has always shown a typical Babinski in both feet. During other febrile paroxysms extension was continually present, proving that the temperature and the slight delirium present each time bore no relation to the form of plantar reflex. Over one one-and-one-half-hour period with a temperature of 95.8°, marked jaw-jerk, spinal epilepsy, spastic knee-jerks, ankle- and patellar-clonus were present.

In this case the complete absence of history and physical signs of organic nervous lesion make us consider whether the typical Babinski reflex may not occur with a normal spinal cord. Dr. Burr tells me that he has rarely found constantly present ankle-clonus and exaggerated knee-jerk in phthisis. These two symptoms have been practically absent in this case, therefore we have to do either with a case in which there is present some tubercular process involving directly or indirectly the spinal cord, and in which there is no other clinical evidence of such a process than the Babinski reflex, or else with an anatomically normal spinal cord showing an anomalous plantar reflex.

THE PLANTAR REFLEX IN CHILDREN.

It is difficult to determine whether the plantar reflex is present in children under one year old, because, without any external stimulus, their toes and feet are almost always in movement. I have examined the same babies on different days, usually with contrary results, and I have observed the same fact in comparing my notes with those of Dr. Burr in cases examined by him the same day. One baby, two weeks old, examined while asleep, showed a typical extension of all the toes as found in the adult. Another baby, also asleep, showed usually typical adult extension. The following percentages are those obtained from tests on fifty babies under one year old:

Extension usually of all toes	50 per cent.
Flexion usually of all toes	22 "
Irregular flexion and extension	20 "
Feet held too stiffly to obtain reflex	8 "
Absence of motion	2 "

In fifty healthy children between one and twelve years of age, typical extension was never found constantly present; in two cases,

one of sixteen months and the other two years, extension usually occurred in both feet. In four cases, two of eighteen months, one of two years, and one of six years, there was irregular flexion and extension. With the exception of this last case neither typical nor irregular extension was observed in children over two years old examined while awake. One child of six years, who, when awake, had active flexion of all the toes in both feet, when asleep showed constant moderate extension of the small toes and doubtful extension of the big toes in response to irritation of the sole or any part of the leg or thigh. In three other children flexion was equally or more pronounced during sleep.

Of the 213 cases of disease of the nervous system with no demonstrable lesion of the pyramidal tract, and under which are included tabes dorsalis, Friedreich's ataxia, paralysis agitans, progressive muscular atrophy, multiple neuritis, epilepsy, imbecility, primary neuritic atrophy, chronic chorea, hydrocephalus, monoplegia, cerebral tumors, cerebellar tumor, brachial neuritis, lead palsy, post-diphtheritic paralysis, neurasthenia, senility, uræmia, Raynaud's disease, cerebrospinal syphilis, cerebral syphilis, toxic ataxias, exophthalmic goitre and hysteria, flexion was present in 167 cases, or 78 per cent. In 38 cases, or in 13 per cent., there was no motion In 5 cases there was constant extension in both feet; in 1 case there was constant extension in one foot, and in 2 cases irregular flexion and extension in both feet. Of the 5 cases with constant extension, 4 were tabetic and 1 neurasthenic, with the following histories:

CASE I.—Tabes. W. B., aged forty-nine years; moulder; white; admitted to nervous wards December 6, 1898, complaining of weakness in legs. Gave history of constant pain in head with increasing deafness for ten years past. Four years ago developed pain and dragging of left leg. and shortly afterward of right leg. Three years ago suffered from a transient diplopia, and since then from girdle pains, acute retention of urine and obstinate constipation. Entrance examination showed a white male, very deaf, with Argyll-Robertson pupils, ataxia of arms, station and gait, absence of knee-jerks and plantar reflex, normal sensation. Since then there is a record of repeated attacks of acute retention of urine, of trophic ulcers on feet and of increasing ataxia. Upon my examination of him in February, 1900. he complained of constant pain in head. He was very deaf, apparently demented, almost helpless from ataxia, Argyll-Robertson pupils were present and the knee-jerks were absent. On the right, stroking the sole produced constant weak extension of all the toes; on the left, stroking sole caused irregular flexion and extension of all the toes. Six weeks later extension was constant in the first, second and third toes of both feet. The patient now failed more rapidly

mentally and physically, and for two weeks before death had marked extension of all toes in both feet.

CASE II.—Tabes. W. S., aged twenty-one years; male; white; admitted to nervous wards June 8, 1898, complaining of pains in head and of nervous spells. No venereal history. Could not walk until three years old, and legs were always weak. One year ago began to notice failing vision and shooting pains in legs. Entrance examination showed a moderately well-nourished white male, about twenty-one years of age; facial expression, dull. Eyes: pupils large, unequal, no light reaction, feeble response to accommodation, slight nystagmus on fixation, extra-ocular motion fair, simple atrophy of both optic nerves; tongue flat, flabby and slightly tremulous; knee-jerks absent both sides, no ankle-clonus; moderate ataxia of station and gait; sensations delayed to touch- and pain-tests over arms and legs. After a history of repeated attacks of laryngeal crises, and increasing dementia, he was transferred to the insane department. Examination, February, 1900, showed patient almost blind, Argyll-Robertson pupils, bedridden, demented, knee-jerks absent. On stroking the soles, active extension of all the toes, most marked in big toes, was produced in both feet.

CASE III.—Tabes. L. S., male; white; aged sixty-two years; admitted to nervous ward May 28, 1896. Two years before admission developed shooting pains in legs and bad feeling around the waist, and later vertigo. Entrance examination revealed a very deaf, poorly-nourished German with Argyll-Robertson pupils, absent knee-jerks, ataxia of arms, station and gait, and some hyperæsthesia of legs. On examination, September 24, 1900, patient complained of pain in legs and attacks of vomiting. He was very deaf, apparently demented; speech feeble, slow and indistinct; Argyll-Robertson pupils were present; there was moderate ataxia of arms, station and gait; the biceps-jerks were active, and the knee-jerks were absent; plantar irritation on right caused active but limited extension of big toe, with no motion in small toes. On the left there was marked extension of the big toe, with indefinite flexion of small toes.

CASE IV.—Tabes. M. F., aged fifty-five years; male; white; admitted to nervous ward November 11, 1898, complaining of pain in left side and back. He has since shown constantly moderate ataxia of arms, station and gait, Argyll-Robertson pupils, absent knee-jerk, and has complained much of pain in left side and shooting pains in legs. Recent examination showed fair mental condition, slight tremor of palm and hands, and repeated tests have shown extension of the big toes with no motion of the small toes in both feet.

In the cases of tabes examined by Babinski, Van Gehuchten and Collier, the extensor response was never met with. Babinski[1] reports a case of tabes, combined with paresis, in which there was typical extension in both feet. Three combined tabes-paresis cases in my own series showed no plantar reflex. Three of the tabes cases above mentioned in detail were undoubtedly demented, yet this fact alone does not seem a sufficient explanation for the presence of the Babinski phenomenon, because in 72 cases of dementia and 28 cases of paresis the extensor phenomenon was never present.

[1] La Sémaine Médicale, Juillet 28, 1898.

CASE V.—Neurasthenia. O. N., male; white; sixty-two years old; admitted to nervous wards December 14, 1899, complaining of general weakness. Examination showed memory, speech, eyes, station, gait and gross strength normal, knee-jerks absent. Plantar irritation caused active extension and separation of small toes on the right; on the left there was moderate extension of the large toe and indefinite extension of the small toes. Repeated and recent examination confirm the above statement.

CASE VI.—Multiple alcoholic neuritis. O. L., male; white; aged forty-three years. Eyes normal, limbs much emaciated, marked diminution of pain and thermal sense over limbs and anterior body, knee-jerks absent. On the right, stroking sole caused good plantar flexion; on the left there is usually slight extension of all the toes, never flexion.

In 85 epileptic cases, flexion was present in 83. In 2, no motion. In a number of examinations during and after a convulsion, while coma was present, flexion was present in all as soon as the spasm had relaxed, except in the following case:

W. W., male; white; aged sixty-four years; history of twelve epileptoid convulsions during past four years. Examination showed feeble-minded, passive, well-nourished male. Gross strength normal, reflexes active throughout; station, gait and sensation fair. Plantar irritation caused fair flexion of all the toes on both feet; no symptoms of cerebral tumor. Patient was found unconscious, with stertorous breathing, by nurse; ten minutes later, when I saw him, he was still unconscious, the head was retracted, limbs rather stiff, biceps- and knee-jerks were active, no ankle- or patellar-clonus. Plantar irritation caused active extension of the big toes, with no motion in small toes on both sides. Seven minutes later he developed flexion of all the toes on right foot, and twenty-five minutes later, absence of motion or indefinite flexion in the left foot. The knee-jerks were slightly increased during the attack. The following day, plantar irritation caused good flexion of all toes on both feet.

Dr. H. O. Shiffert, a Blockley colleague, tells me that in one patient during an uræmic convulsion he obtained a typical extension of all the toes on both feet. Later I examined the same patient while in a state of coma and found normal flexion in both feet. I have examined seven uræmic cases in the state of coma. In six flexion was present, and in one there was no plantar reflex.

HEMIPLEGICS AND DIPLEGICS.

In 118 hemiplegics extension was present on the paralyzed side in 57 per cent.; flexion of all the toes in 26 per cent.; flexion only of the small toes, 4 per cent.; irregular flexion and extension, 5 per cent.; no motion, 8 per cent. On the opposite side extension was present in 9 per cent.; flexion of all the toes, 72 per cent.; flexion of the small

toes, no motion in the big toes, 4 per cent.; irregular flexion and extension, 5 per cent.; no motion, 10 per cent.

The duration of the hemiplegia varied from six hours to thirty-nine years, and, apparently, bore no direct relation to the time of appearance or the form of the Babinski reflex.

CASE I.—L. B., male; black; aged forty-five years; history of complete hemiplegia of six hours' duration. On admission there were spastic knee-jerk and absence of plantar reflex on paralyzed side. These conditions remained the same until six weeks later. Fourteen weeks later I again saw the patient, and at that time the knee-jerk was weak, and stroking sole caused moderate, slow extension of all the toes on the paralyzed side.

CASE II.—M. S., white; female; aged forty-three years; two weeks before admission developed partial paralysis of right face. Twelve hours before admission, patient was found unconscious with stertorous breathing, difficult deglutition and loss of power in right arm, with temperature, pulse and respiration, respectively, of 102°, 80 and 32. On admission examination showed a well-nourished white female, unable to talk, but conscious of all that was said; some drooping of right face; eyes showed no deviation, paresis, ataxia or nystagmus; right hemianopsia was probably present; tongue moist, clean, lay motionless in floor of mouth; pulse, 72, full and regular; heart-sounds obscured by noise of breathing; lungs resonant; breathing vesicular, accompanied by large, moist râles; abdomen showed nothing abnormal. Extremities were rather rigid, especially the right arm, in which no motion, except slight flexion of fingers, was possible; other limbs were well moved. Reflexes were all spastic; patellar-clonus both sides; ankles stiffened by old joint lesion, and showed no clonus. There was normal plantar flexion in both feet. Sensation was normal; urinary and fecal incontinence; urine examination, negative. Thirty-six hours after stroke the reflexes were no longer spastic, though large; no clonus. Plantar irritation caused the extension of all toes on the right, and flexion of all toes on the left. Forty-eight hours after stroke the right leg had become flaccid and powerless; knee-jerks were absent; extension of toes persisted on the right, and flexion on the left. Sixty-seven hours after stroke the knee-jerks were still absent, and extension was present in both feet, more marked in right. Until death, which occurred four days later, the knee-jerks remained absent, extension of all toes persisted in both feet, and the paralysis remained limited to the right side of body, limbs and face. Post-mortem revealed congestion of cerebral meninges; ventricles contained an increased amount of clear serous fluid; small hickory-nut-sized area of softening in the right corpus striatum; kidneys showed no gross lesion.

CASE III.—M. J., female; white; aged sixty-two years; always healthy. The day before admission suddenly developed double vision and a sense of nausea. Entered hospital with incomplete external and internal ophthalmoplegia. Gait and station poor, falling always toward left if not supported; marked jaw-jerk, biceps-jerk and knee-jerk. Plantar irritation caused extension of all toes on both feet. Three weeks later complete right hemiplegia developed, but had caused no change in the reflexes described above when the patient was examined five days later.

In seven diplegics, extension was present in both feet in three cases, irregular flexion in three cases, and weak flexion in one.

THE PLANTAR REFLEX IN SPINAL CORD DISEASE WITH LATERAL TRACT INVOLVEMENT.

In this series are included spinal syphilis, cerebrospinal syphilis, myelitis, compression myelitis, traumatic myelitis, syringomyelia, multiple sclerosis, amyotrophic lateral sclerosis, diffuse spinal sclerosis, ataxic paraplegia, spastic paraplegia, spastic monoplegia, meningomyelitis, and infantile paraplegia.

In the 62 cases of this class, extension was present in 63 per cent.; flexion in 26 per cent.; no motion in 11 per cent.

The following is a case, physiologically at least, of total transverse lesion of the spinal cord.

M. M., aged forty-five years, female, white, was admitted complaining of loss of power in legs. Six months ago was operated on for carcinoma of the breast of eighteen months' duration; noted return of nodules in scar two weeks ago. Ten days before entrance developed paræsthesia, shooting pains, some loss of sensation and weakness in legs; three days before admission developed complete paraplegia and loss of sensation in legs, with incontinence of urine and fæces. Examination showed moderately emaciated white female; eyes, normal; heart, lungs and abdomen showed no gross lesion. There was a small, hard nodule in the cicatrix of excision of left breast, and a few hard bodies could be felt in left axilla; there was some prominence of the seventh, eighth and ninth dorsal vertebræ. The extremities were much emaciated; reflexes of arms active; the legs were completely paraplegic, and there was complete loss of sensation over legs and on body up to one inch above umbilicus; knee-jerks were absent; sphincters were completely incontinent. Stroking the soles, or irritation of any part of the legs, produced usually quick flexion of legs and thighs, associated with dorsal flexion of the ankles and irregular extension of the big toes, more marked on the left.

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THE USE OF METHYLENE BLUE AS A SEDATIVE.

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AND
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The use of methylene blue in the diagnosis of nephritis and in distinguishing the different forms of this disease has been carefully studied by several clinicians. Cabot and McGirr, in an article in the *St. Paul Medical Journal* for December, 1898, report a series of valuable observations upon the subject. Ehrlich and Guttman ascribed to methylene blue a chemical affinity for the axis-cylinder of nerves, and reasoned from this that it must exert some definite action upon the nervous system, and proposed its use as a sedative in nervous or mental excitement. But Combermarle's investigations proved that the chemical affinity existed only for dead tissue. Bourge demonstrated its power as a vaso-constrictor, and no doubt it is to this property that it owes any sedative action it may have. Bodoni, working in Morsilli's clinic, while giving the drug to differentiate the forms of nephritis, observed that each patient was depressed while taking it, and proposed its use in mania, puerperal mania, and the excited states of paretic dementia. He accordingly tested it in fourteen cases, of which he gives a detailed report in the *Klinisch Therapeutische Wochenschrift*, No. 21, 1899. His cases were mania with frenzy, having a basis of degeneration, two cases; mania with fury, mania simple, mania periodic, mania congestive (alcoholic), each one case; and mania chronic, two cases; also melancholia periodic with maniacal outbreaks, paranoia with delirium, dementia secondary, the insanity of hystero-epilepsy and puerperal mania, each two cases. Each patient was so satisfactorily subdued by it that Bodoni proposed to place methylene blue in the list of sedatives with trional, sulphonal and chloralamide and like drugs. The drug has now been used in twenty-two cases in the insane and detention wards of the Philadelphia Hospital, and while the results were not so uniformly good as in the cases reported by Bodoni, it has led to the hope that a greater number of trials may

demonstrate that in particular cases the value of the medicament as a sedative may be established. Many of the histories following are given more fully than is the custom in such reports, in order that a clear picture of the mental condition may be understood by the reader. The development of abscesses was an annoying and painful complication in the first cases in which the drug was used by the hypodermic method. Although the usual antiseptic precautions had been observed, it having been forgotten that methylene blue often contained many bacteria. Boiling the bottle containing the solution prevented the abscesses afterward. A further trial is being made with the drug, the results to follow in a subsequent report.

CASE I.—B. K., female, aged forty-six years; married; Russian; nine children, the oldest thirty years and the youngest twelve years; admitted July 31, 1899, being her third admission, the first in 1894 and the second in 1897. She was wildly maniacal, scolding in a shrill voice, hostile, abusive, profane and obscene, scratching and spitting upon any one coming near her, and most destructive, tearing her clothing and bedding into fragments so that it was impossible to keep her clad or covered. She refused medicine and food, declaring that it was poison and horse blood.

There was constant auditory hallucinations, excitement and insomnia. The physical complications were an unhealthy fistula in the median line of the epigastrium (the sequel of an operation for hernia two years before); a severe gastro-enteritis and a severe grade of nephritis; a temperature ranging from 66° to 76° and irregular. The temperature was no doubt toxic aud due to the discharging fistula or to the gastro-intestinal condition. The urine analysis 1032 acid, much albumin, many wide and narrow hyaline and granular casts, and the amount voided in twenty-four hours was but fourteen to twenty ounces.

From August 31st to September 12th the usual sedatives were used by the hypodermic method and by enemata, but failed to control her restlessness or to produce sleep, and she was rapidly losing flesh and becoming exhausted. She was nourished by enemata. Three Widal tests were negative.

At 12.30 P.M., August 12th, one grain of methylene blue, in aqueous solution, was injected deeply into the gluteal mass by means of an anti-toxin syringe. After 3 P.M. the patient began to grow quieter and at 5.30 P.M. answered a few questions respectfully, the first since her admission. Urine voided at 5.40 P.M. was of a dark bluish-green color and showed no changes in character except that some of the hyaline casts were lightly stained green. She slept six hours during the night, being quiet when awake until 7 A.M., when she became restless and talkative. One grain was injected at 9 A.M. and another at 6.30 P.M. She was quiet after 11.30 A.M., and took some milk and vino kolafra. She remained awake all day, but slept seven and one-half hours the following night. Each of the next six days one grain was injected at 9 A.M. and at 6.30 P.M., with most satisfactory results. She took food and medicine well, was quite pleasant to the nurses and interested in the other patients about her, but was still hostile to her husband and children, scolding and ranting at them for "trying to put her away."

Two days after the first injection an induration developed at the point of puncture, and at the side of each subsequent puncture a like node formed. They were very dense, pale-red and neither painful nor tender. They were painted with iodine and ice was applied. The redness faded and they diminished in size, but remained when she was paroled three months later.

On account of the indurations the injections were discontinued from August 20th until August 24th, when she again became restless, abusive and destructive.

From August 24th until September 2d, morning and evening, doses were continued, the good effects being apparent after the first dose. She was then quiet, docile and cheerful. The temperature was normal after August 22d. She took food and medicine well and permitted antiseptic dressing of the fistula, which soon became healthy, and was entirely healed when she left the hospital.

Every known precaution was taken in giving the injections and in caring for the points afterward, but nodes now studded the buttocks and thighs and varied from 3 to 5 cm. in diameter; they were anæsthetic and gave the patient no inconvenience whatever. On September 5th she showed signs of restlessness, and one grain was injected daily until September 19th. The quieting influence of the drug was manifest after the first dose. From this time she was kind and helpful in the ward, grateful for what had been done for her, and she seemed to have lost all of her delusions except those concerning her husband. This condition remained unchanged when she was paroled. She has since visited the institution and seems quite rational.

CASE II.—B. W., female, aged thirty-two; single; Irish; domestic; admitted November 28, 1899; case of acute mania complicated by severe nephritis, the urine containing albumin and hyaline granulas and blood-casts, the amount voided daily being but fourteen to seventeen ounces. She was greatly emaciated and very restless. One-grain doses of methylene blue injected into the thigh daily produced a quieting and calming effect which was noted after the second dose; but five days after the administration a deep-seated abscess developed at the point of injection in the left thigh, and on this account the drug was discontinued. The abscess began as a dense induration similar to those described in Case I.

The habits of this patient were most uncleanly and undoubtedly the puncture-wound was infected by herself. The abscess healed slowly; the patient responded poorly to other sedatives and continued disturbed until she died of her nephritis, three months later. In this case the urine voided six hours after the first dose was colored dark bluish-green, and thirty hours after the last dose was free from color.

CASE III.—L. G., female, aged twenty-nine years; single; Russian; admitted August 27, 1889; a case of recurrent mania, the outbreaks being of six to fourteen days' duration and occurring always at the menstrual epoch, but not at every period. One-grain doses were administered hypodermatically for three days, and then the same dose twice daily for three days, without effect. The indurations formed as in the other cases, but did not suppurate. At the end of two months they had disappeared.

CASE IV.—L. D., colored, female, aged forty-four years; married; admitted August 10, 1899; a case of chronic mania complicated by nephritis and phthisis pulmonalis. She was always noisy, destructive and hostile, and from the time of her admission had been a most troublesome patient. One-grain doses of methylene blue, administered twice daily, produced a marked quieting effect, shown after the second dose; but on the fourth day an abscess began to form, and the injections

were discontinued on the seventh day of treatment. The abscess was a very small and superficial one, and promptly healed. Accurate record of the color changes in the urine could not be kept in this case on account of an obstinate diarrhœa.

CASE V.—M. B., female, aged forty-three years, a Philadelphian, married and a patient of the insane department almost constantly since July 16, 1887, being a case of periodic mania complicated by nephritis. After a short parole to her family she was returned in a greatly excited condition, talking rapidly and constantly, incoherent and restless. One-grain doses of methylene blue, given twice daily, produced a prompt quieting effect, apparent after the third dose, but on account of the formation of an abscess in the thigh the drug was discontinued on the fifth day. Until after the third dose the patient would not permit the protective dressing to remain over the point of puncture, and her habits were very uncleanly, which would account for the infection.

CASE VI.—H. O., female, aged forty-one years; Irish; single; admitted October 31, 1896; a case of recurrent mania of twenty years' standing, she having spent almost this entire period in the hospital. Her attacks were always most violent and of several weeks' duration, and occurred at intervals of from five to seven months. After having been quiet and industrious for several months she was paroled to her family January 1, 1900. Four days later she was returned in a wildly maniacal state, screaming, abusive, assaulting, tearing her clothes and hair, profane and resistive. January 18, 1900, the daily administration of one-grain doses of methylene blue was begun and continued for two weeks, producing a prompt controlling influence, which was apparent after the third dose. Until the present time there has been no return of her mania, this being an unusually long interval.

CASE VII.—E. S., female, aged thirty-three years; married; Russian; admitted December 28, 1899; a case of chronic mania. She was treacherous and cruel, with almost daily outbreaks. One-grain doses of methylene blue were administered twice daily; they controlled her frenzy, but she continued to be stubborn and resistive. The drug was given twenty-two days in this case without any untoward symptoms, the patient steadily gaining flesh and color, and an obstinate insomnia was broken up.

CASE VIII.—S. H., female, aged twenty-seven years; married; Russian; admitted April 7, 1900; a case of puerperal mania of six months' duration. She refused food, was restless, sullen, obstinate and resistive, and had most uncleanly habits. One grain of methylene blue was administered twice daily for four days, when a large bleb formed at the site of one puncture. After the second dose the patient relaxed and became more tractable and ceased to scream, but continued to be sullen and morose.

CASE IX.—A. C., female, aged forty-three years; widow; Irish; admitted March 2, 1899; a case of mania epileptica and a very vicious, treacherous and dangerous patient when excited. Her convulsions, fortunately, were infrequent, but they were always followed by violent mania, which persisted for several weeks. One-grain doses of methylene blue, given twice daily, produced a calmative effect, noted after the second administration, and after the fourth she was kind and cheerful. From May 2 until May 27, 1900, one-grain doses were given daily, with several two to four days' intermissions, when the patient always became restless and noisy, but grew quiet when the drug was again given. Indurations appeared at the points of puncture, but they were small and soon disappeared. At this time it was decided

to change the mode of administration—partly on account of the indurations and partly to test its efficiency when given by mouth. A capsule containing two grains of methylene blue with aloin was used. In this and all other subsequent cases the capsule was given first twice, then three times daily from May 28th until June 8th. May 30th she had a convulsion, followed by a half-hour's period of excitement, which was much milder than former attacks. She steadily gained flesh and color, and no unpleasant symptom was noted at any time.

CASE X.—P. M., female, aged thirty-five years; married; admitted October 28, 1898; a case of recurrent mania complicated by severe nephritis. The two-grain capsules administered three times daily for four days were without effect. This patient, however, was not quieted by any other sedative except a combination of morphine and hyoscine, and continued disturbed until her death, two months later, of nephritis.

CASE XI.—A. S., female, aged thirty-seven; married; admitted May 23, 1899; a case of recurrent melancholia with periods of agitation occurring at each menstrual epoch and requiring bed-treatment for about one week, when she was noisy, talkative, incoherent, sleepless, untidy and vicious. When in an excited state (June 9, 1900) she was given the two-grain capsules three times daily. The following day she was much quieter, and the third day was out of bed and usefully employed. No unpleasant results followed, and the capsules were continued over the next period, which passed with one day's bed-treatment, with little agitation.

CASE XII.—J. K., female, aged forty-five years; married; Hungarian; admitted May 30, 1900; a case of acute melancholia with maniacal outbreaks. After a period of depression she suddenly became noisy, restless and hostile. This was on July 11, 1900, and the two-grain capsules of methylene blue were given three times daily until July 17th, when she complained of nausea. She was quiet after the first dose, and after the third took a pleasant interest in her surrounings. She developed dysentery (then prevalent in the hospital) on July 18th, and the nausea may have been an initial symptom of the dysentery and not due to the methylene blue.

CASE XIII.—C. J., female, aged fifty-six years; married; German; admitted February 5, 1898; a case of circular insanity of twelve years' standing and complicated by nephritis. May 18th she became excited and troublesome, talking incoherently, scolding, and pacing back and forth in the wards. She was given a two-grain capsule of methylene blue at 7.30 P.M., was quiet at 9 P M., and slept eight hours the following night, but was noisy after waking. The capsules were continued from May 18th to June 6th, with most satisfactory results. She was quiet and docile when under the influence of the drug, but became restless when it was withheld. On June 6th she complained of vertigo and the treatment was suspended until August 4th, when she again became restless and excited, and the capsules were given. While taking them she was somewhat disturbed on the fourth day, was dull and listless on the fifth day, bright and cheerful on the sixth day, but talked more than was natural and was incoherent at times. After that time she was out of bed and usefully employed in the ward, where she now is a most faithful helper. In this case two attacks have been aborted. After each of the two periods of excitement some depression was noted, but it was very mild. Former cycles, which were frequent and most severe, had lasted from six to nine weeks, with a maniacal stage of about two weeks, when in her excitement she would beat herself, refuse food, and was very noisy, especially at night.

CASE XIV.—R. D., male, aged forty years; married; American; bookkeeper;

admitted April 4, 1900. History of profound melancholia of several months' duration with an active suicidal impulse. He believed that it was said that he had signed certain papers, causing a loss to his employers of many thousands of dollars, and that he had benefited by the irregular transaction. He had the most woebegone expression, was resistive, refused food, refused to talk or to walk. If any attempt was made to force him to dress or leave his bed or walk he would grow angry and shout and swear, and if still further forced (without violence, of course), he would grow very violent and strike the nurse. He refused food and medicine, and for weeks was fed with either the nasal or oral tube. For four weeks after his admission he didn't take a drop of water or other liquid voluntarily. He suffered greatly from insomnia. He lost flesh rapidly and it was feared he would die from inanition, as the heavy tube-feeding did not supply sufficient nourishment. Sedatives by the hypodermic method, bowel and tube, made no impression upon the mental condition. After six months' treatment all other medication was stopped and he was given one grain of methylene blue in aqueous solution by the hypodermic method. The next day he was given one grain in the same manner night and morning, and so on for the next six days. On the morning of the third day of the new treatment he drank a glass of milk and a glass of iced water. On the next day he ate for his breakfast a bowl of cracked wheat with milk and drank a cup of coffee. At the end of six days the injections of the methylene blue were reduced to one a day of one grain each. He was now eating his meals regularly, was sleeping well, was respectful in conduct, had grown much less restive, and the terrible agonizing expression of the face with the large number of transverse wrinkles of the forehead had disappeared. At the end of another six days he was able to sit up and dress himself and would answer a few questions. The injections were now given every other day for another six days, when they were discontinued. In two months from the time of the first injection of the methylene blue he was eating regularly, sleeping well, talked a little and fairly rational with his wife, but more rationally with his special nurse, walked each day in the yard and occasionally looked at a daily paper. He also took his medicine regularly and without opposition or a sense of fear. The change in his condition was simply marvellous, and an early recovery was anticipated. But at the end of the tenth week he had a severe maniacal outbreak, shouting, cursing, striking, and pulling his hair and mutilating his face, and demanding his immediate discharge, refused food and medicine, and was wakeful for two nights, when he was again given a hypodermic injection of methylene blue in aqueous solution of one grain each morning, and at the end of seven days he was again quiet and had returned to the mental state he was in before this last outbreak. He was now given the methylene blue in capsules of one grain each, night and morning for three weeks, after which it was stopped. He is now quiet and orderly, eating, sleeping and talking well, and seems to have begun a permanent convalescence. The urine always, within four hours of an injection or five hours of a capsule, showed the staining of the drug, which only gradually disappeared not altogether for a week or more after the drug was discontinued.

CASE XV.—C. L., male, aged thirty-one years; clerk; admitted September 12, 1900, to detention ward, in condition of delirious mania, resistive, spitting at physician and nurse, refusing food and liquids and tossing and shouting constantly. Mouth dry, eyes blood-shot, skin bathed in profuse perspiration. Urine and stools voided in bed. Force required to hold patient for initial injection of

one grain of methylene blue. No effect was noticeable, and in five hours the injection was repeated, at which time it was first noticed that the urine voided was slightly stained. Within a half hour after second injection patient was quiet but still resistive. A quiet, but sleepless, night was passed and once patient took bovinine in hot milk. A third injection of one grain was administered the following morning, twelve hours after the second, which was followed by a restless sleep of four hours, and on awakening answered few questions, protruded tongue, permitted sponging and took a quantity of milk. Towards evening, again became restless and resistive, and had to be held for the fourth injection, which was soon followed by a quiet sleep. Upon awakening was very rational, permitting attention to toilet, and took a fair meal. For the next three days two injections per day, morning and night, were given, and the patient passed into a quiet convalescence, and on the fourteenth day was removed to his home in another State, having had one injection daily until discharge. The history obtained from friends showed the attack to have suddenly followed the loss of considerable property through deception of relative. There was a distant hereditary taint.

CASE XVI.—A. R., female, white, aged twenty-eight years; domestic; admitted to detention ward in condition of violent acute mania. Incoherent, hallucinatory, and delusional. Face flushed, eyes injected, tongue heavy, brown, dry fur, skin dry, bowels costive, bladder atonic, catheters necessary. Urine showed albumin, specific gravity 1028, urates, hyaline casts. Methylene blue in aqueous solution (one grain) injected, and four hours after, no benefit shown, the injection was repeated. At the end of ten hours the maniacal condition had increased, with evidences of exhaustion. Pulse, 120; respirations, 32; temperature, 103°. A third injection was given, and four hours after a fourth, but all without effect. The condition of patient growing more serious, morphinæ sulphas (one-third grain) and hyoscin hydrochloras ($\frac{1}{100}$ grain) were added to the fifth dose of the methylene blue. The combination gave a fitful sleep of two hours, the patient awakening as maniacal as ever. Uræmia being considered the cause of the mania, a free venesection was performed, followed by the injection under the skin of two pints of a normal salt solution, and free purgation continued. After the withdrawal of the blood the mental symptoms were much improved, but the patient continued restless. Six hours after the fifth injection of the medicaments another injection was given, which was followed by a restless sleep of four hours. Upon awakening the patient was greatly exhausted, the pulse reaching 150; the respirations, 40; the temperature, 100°. Stimulants were boldly pushed with injections of strychniæ sulphas; the patient grew less excited and resistive. Ten hours after patient was given another injection of methylene blue and within half an hour was sleeping quietly. It was considered advisable to awaken the patient in three hours for nourishment and stimulants. As soon as taken again went to sleep, and was awakened in four hours for food, quiet but incoherent. Continued much in this condition for three days longer, sleeping quietly greater portion of time. On the seventh day the temperature suddenly arose to 107°, the patient becoming rapidly comatose, death soon following. The urine showed the blue staining within six hours after the initial dose and continued so until death. Autopsy showed parenchymatous nephritis.

CASE XVII.—J. W., white, aged thirty years; American; painter; married; admitted to detention ward September, 1900, in condition of violent acute mania. Unable to secure any coherent statement, continually chattering, flying from topic

to topic and jumping from bed, struggling and gesticulating wildly, refusing food and medicine by mouth or enema. Pupils dilated, eyes staring, face flushed, tongue dry and coated, abdomen distended. Urine voided involuntarily. Impossible to secure temperature, pulse and respirations. At three o'clock he was given hypodermatically methylene blue (one grain), which was repeated in five hours. Slept one hour after second dose, but balance of time noisy, spitting and biting when approached. At seven in the morning, at noon, and at seven in the evening, one grain of methylene blue was again administered hypodermatically. On this evening he accepted eight ounces of milk in which had been placed calomel and bicarbonate of soda, each five grains. Slept portion of the night quietly, and when awake was quieter and more coherent. For the next four days had one grain morning, noon and night, gradually growing more quiet and coherent, and by end of the week was entering mental convalescence. He was discharged in the fourth week fully recovered. The urine became of a dark bluish-gray color eight hours after the first injection, and continued discolored, gradually lessening, for sixty hours after the last dose was given. The sedative result in this patient was the most prompt and decided of any to whom the drug was given.

CASE XVIII.--H. G., negro, aged thirty-three years; native of South Carolina; waiter; admitted July 16, 1899, suffering from dementia paretica with maniacal outbreaks, during which he struck wicked blows and was very destructive. It was only safe to keep him in a ward with other patients by the use of large doses of sulphonal or chloralamid. On October 5th he was placed upon capsules containing one grain methylene blue, with one grain salol, three times daily. The outbreaks became much milder and at wider intervals. The drug exerted no influence over the downward course of the disease, but for the next six months he was a fairly quiet patient. The urine remained stained during the entire course of the drug, but no untoward symptoms developed.

CASE XIX.—B. O. T., white, aged fifty-eight years, American. In hospital five years. Homicidal mania with epilepsy. Every two or three weeks been having violent maniacal outbreaks. For one year been taking a nightly dose of chloralamid (forty to sixty grains), which checked the violence and impulsiveness of the outbreaks to a great extent, and permitted his being in a general ward, but under watchful eye. July 10, 1900, he was ordered methylene blue (one and one-half grains) in capsule with aloin ($\frac{1}{20}$ grain) three times daily. No change was noticed in his disposition for two weeks, from which time until this report, three months after, he had no outbreaks and was of a more pleasant disposition. The urine did not show the discoloration for fourteen hours after the beginning of the drug.

CASE XX.—W. M., male, white, aged thirty-six years; laborer; admitted, 1900, with history of epilepsy of seven years' duration following immediately after attack of typhoid fever; also intemperate. Until September, 1899, had been confused only after convulsions. From this time on was violent and destructive after each paroxysm, lasting for several days. Epileptic paroxysms also increasing in frequency and severity. He was placed in turn upon bromides, sulphonal, hyoscine and chloralamid, without result. In September, placed upon a capsule containing methylene blue (one and one-half grains) with aloin ($\frac{1}{15}$ grain) three times daily. For the month he had but ten paroxysms against an average of thirty the months before, and but one excited spell, and this without the impulsive violence of before, and with a decided improvement in the condition. The further

results must await for another report, as this one goes to the printer. The urine was first stained ten hours after first capsule was taken.

CASE XXI.—G. S., female, white, aged twenty-three years; American; maid; admitted to detention ward with history that for two weeks had been mentally disturbed with alternating periods of depression, with weeping and wailing, followed by attacks of mania. Delusions of poisoning and other injuries by her family. Upon admission incoherent, auditory hallucinations, excited, restless, throwing arms in every way continually, and attempting to jump from her bed, so that restraint was necessary. Two hours after admission was given a hypodermic injection of methylene blue (one grain). No effect observed in four hours, and patient growing more excited and restless, the injection was repeated, and in two hours a third injection, and in four hours a fourth; the patient beginning to show marked signs of exhaustion, a hypodermic injection of morphinæ sulphas (one-third grain) with hyoscinæ hydrochloras ($\frac{1}{100}$ grain), which was almost immediately followed by a heavy sleep. Methylene blue was not again used. The urine of patient was, upon admission, of a heavy color, with a muddy sediment, specific gravity 1030, containing albumin, granular casts and much epithelium. Urine was decidedly stained five hours after first injection, and was clear twenty-five hours after the fourth and last injection. This patient made a slow recovery under the usual treatment employed in the insane wards.

CASE XXII.—S. M., white, aged thirty-three years, married. An elder sister had had an attack puerperal mania. Patient had mild attack of puerperal mania four and one-half years before, fully recovering. Present attack began suddenly by patient declaring she heard her father and mother crying, and asking those about her if they also did not hear them. Declared some one was going to die, and insisted on going home, talked strangely, growing incoherent and maniacal, constantly talking and striking any one coming near her. If offered food or medicine would bite and spit and strike. Insomnia most decided, screaming for hours and hours at a time. Immediately after admission was given hypodermic injection of methylene blue (one grain), which was repeated every four hours for three days without any quieting effect whatever. Exhaustion being imminent, morphinæ sulphas and hyoscinæ hydrochloras were used by the hypodermic method with the result of giving a few hours' restless sleep, but the maniacal excitement being as intense as ever on awakening. The patient suffered from a severe nephritis, and to increase the flow of urine normal salt in pint amounts was injected under the skin every few hours. The patient developed pneumonia, dying on the ninth day after admission, during which time she had had ten hypodermic injections of methylene blue without showing any benefit. She also had four doses of sixty grains of chloralamid on the fifth day after admission, deriving no benefit from the drug.

These were nearly all cases of wild excitement when the drug was used, and in but six did it fail to produce a calmative effect which did not resemble the sedative action of other drugs, but seemed rather a natural quietude—the patients were relieved of excitation, but without dullness or hebetude. The effect was noted three to four hours after a dose was given and lasted from fifteen to twenty hours.

No depression resulted at any time except in Case XIII. In each

case the patient slept well at night, but in none did it produce sleep in daytime. In but one case was there any unpleasant symptom (vertigo) which could be surely attributed to the drug, although in Case I it was given hypodermatically for fourteen consecutive days, and in other cases, forty-two, twenty-nine, twenty-two, twenty-one, and fifteen days, by mouth, proving that, at least, it is a safe drug. Only two of the patients (Cases III and VII) could be considered healthy, and each improved physically while taking it.

The records of cases IV and XII were incomplete on account of diarrhœa. The time at which the color appeared and disappeared in cases III and VII were so uniform with those in which there was nephritis that careful uranalyses were made, but they were always negative. Cabot gives the average time at which the color disappeared as sixty-four hours; in these cases it was seventy-two hours and fifty-six minutes.

The quieting effects of the drug by stomach administration, as shown in Cases XVIII, XIX and XX, followed without derangements of the gastro-intestinal canal, and if future administrations confirm these results, a most valuable addition has been discovered for controlling many annoying chronic incurable mental cases, whose excitement is a source of so much annoyance in hospitals for the insane.

The results from treatment in these few cases entitle the drug to further study, and give it a place in the ever-growing class of sedatives. It is now being tested in additional cases in this hospital.

The changes noted in the urine, with the complications in each case, are summarized in the following table:

	Time after first dose when color appeared. Hrs.	Min.	Time after last dose when color disappeared. Hrs.	Physical Complications.
Case I.—First administration . .	4	50	72	Nephritis, fistula and gastro-enteritis.
Second " . .	8	30	80	
Third " . .	7	15	80	
Case II.	6		30	Parenchymatous nephritis.
Case III.	5		72	None.
Case V.	6		61	Interstitial nephritis.
Case VI.	7		68	Nephritis.
Case VII.	10		63	None.
Case VIII.	2	40	63	Gastro-enteritis.
Case IX.	8		78	Nephritis.
Case X.	4		98	Nephritis.

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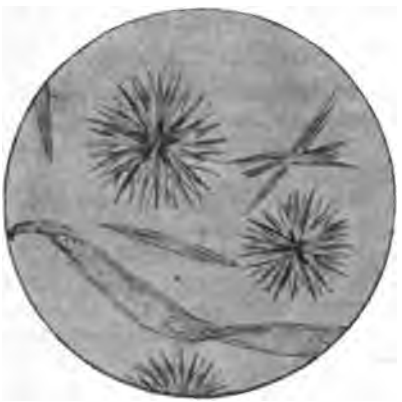
No depression resulted at

	Time after first dose when color appeared. Hrs. Min.	Time after last dose when color disappeared. Hrs.	Physical Complications.
Case XI.	4	92	Gastritis.
Case XII.	8	Not recorded.	Gastritis.
Case XIII.—First administration,	4	68	Nephritis.
Second "	8	96	
Case XIV.	4	104	None.
Case XV.	5	50	Gastric Catarrh.
Case XVI.	6	Death.	Nephritis and Uræmia.
Case XVII.	8	60	None.
Case XVIII.	12	60	Paresis.
Case XIX.	14	80	Epilepsy.
Case XX.	10	Still using.	Epilepsy.
Case XXI.	5	25	Nephritis.
Case XXII.	8	30	Puerperal.

SOAPS OF LIME AND MAGNESIA IN URINE.

By GEORGE E. PFAHLER, M.D.,
ASSISTANT CHIEF RESIDENT PHYSICIAN.

This sediment being found rarely, but in a variety of diseases, may have some clinical significance. It occurs as colorless, highly refractive crystals, which in form resemble tyrosin, but which possess distinctive characters of their own. They are larger and the individual spicules are more tapering than those of tyrosin. The accompanying illustration will show their form. They are soluble in ascetic and hydrochloric acids, in ammonia, and slowly soluble in water.



Soaps of calcium and magnesium from urine of a case of abscess of liver, also found in acute mania and empyæmia. By Dr. Pfahler.

I have found these crystals in the feebly-acid urine of three cases in considerable numbers. The first case was one of acute mania. The urine was obtained by catheter during an outbreak, and after standing a short time showed these crystals.

The urine was examined repeatedly afterward, but never again during an outbreak, and failed to show this sediment.

The second case was one of abscess of the liver. The patient's temperature and general condition were those of a septic process.

The urine was passed normally, and after a few hours showed these crystals. The patient was removed from the hospital a few days later, so that this was the only specimen obtained showing the sediment.

The third patient was one of cocaine poisoning.

The specimen showing these crystals was the first obtained after the profound toxic symptoms. They were not found in subsequent specimens.

V. Jaksch has had the opportunity of examining these crystals only once, finding them in considerable abundance, and in the feebly-acid urine of a woman with severe puerperal septicæmia.

The toxic nature of the above cases may have some bearing upon the formation of these crystals, and justify my making this brief report.

TWO CASES OF TRAUMATIC TETANUS. DEATH.

By D. E. HUGHES, M.D.,
CHIEF RESIDENT PHYSICIAN.

Ten cases of acute traumatic tetanus have been admitted to the wards of the hospital at irregular intervals since 1890. Nine have died. One recovered under the use of hypodermic injections of cocaine hydrochlorate. I have now treated successfully three cases of traumatic tetanus with cocaine by the hypodermic method, the one just mentioned and two cases in private practice. Of the nine cases dying in this hospital two were treated with the tetanus serum and one with the serum and carbolic acid by the hypodermic method, as suggested by Baccelli. The third case mentioned was treated by the serum, but under the direction of another member of the hospital staff, and is not reported in this paper.

W. W., admitted October 1, 1899; white; aged thirty-four years; laborer. Dr. Stackhouse, Interne. A well-developed man and healthy previous to September 22, 1899, when his right foot was punctured by a nail. No attention was paid to this injury, the wound being slight. On September 28th he noticed his jaw "feeling tired." On the 29th he began having pains in the muscles of his back, which felt "as if they were being tied into a knot."

October 1st.—He was admitted to the hospital just eight days after the injury, complaining of pain in his back and stiffness of his jaw. Examination showed a bleb covering the site of the puncture which contained a small amount of purulent material. The jaws were very firmly fixed, it being only possible to force the mouth open one-fourth of an inch. The head freely movable. The back somewhat stiff, but the patient able to walk. The reflexes are increased generally, the knee-jerks being snapping in character. The skin sensitive over the back, abdomen and thigh. Temperature, 99°; pulse, 84; respirations, 24.

October 2d.—Is not so well this morning; the stiffness of the back is increased so that when an attempt is made to raise the patient he slides toward the foot of the bed. During the day he received by subcutaneous injection three grains of acidum carbolicum (2 per cent. solution) and 20 c.c. anti-tetanic serum. This evening his general condition seems better, his pulse is full and regular and his respirations free; but his back, which last night was free, is now slightly arched.

October 3d.—To-day patient was given subcutaneously six grains acidum carbolicum (2 per cent. solution) and 60 c.c. anti-tetanic serum. Seems more comfortable this evening, but there is an increase in the arching of the back and the jaws are very rigid. Legs also very rigid this evening. Also very sensitive to

external impressions, an unexpected voice, a heavy footstep or moving any article of furniture across the ward bringing on spasms. The paroxysms are increased in frequency and duration when patient lies on his side. He twitches a great deal, even in his sleep. One stool during the day but voided no urine, twelve ounces, however, being drawn by catheter. Urine showed few hyaline and granular casts. There is no twitching of the muscles of the face and neck; the head, however, is thrown back, as it seems to relieve his locked jaws and his breathing. His mind is perfectly clear. He takes nourishment well.

October 4th.—During the day administered eight grains acidum carbolicum and 60 c.c. anti-tetanic serum. Bowels have not moved for twenty-four hours. Voided twelve ounces of urine in bed. Sleeping heavily greater part of the day and took but little nourishment. Temperature has been subnormal past twenty-four hours. Body and face covered with heavy perspiration. Paroxysms increasing in number and severity, greater part of day occurring every five or ten minutes.

October 5th.—During day received twelve grains of acidum carbolicum and 40 c.c. anti-tetanic serum. Early this morning a protracted convulsion lasting ten minutes. After this the opisthotonus was less marked and he could open his mouth fully one-fourth of an inch. He voided fifteen ounces of clear urine of a specific gravity 1024. Toward the latter part of the day he was mildly delirious. Temperature varied between 97° and 100° during the day.

October 6th.—During day received twelve grains acidum carbolicum and 40 c.c. anti-tetanic serum. This morning a convulsion lasting several minutes, but during the day less arching, and the mouth could be opened voluntarily one-fourth of an inch. During the greater portion of the day delirious. Urine only ten ounces, but clear. Temperature varied between 97° and 100° during the day. Took nourishment well.

October 7th.—Received during the day twelve grains acidum carbolicum and 40 c.c. anti-tetanic serum. The paroxysms are growing more frequent and severe, having had one during the day which lasted one-quarter hour. The opisthotonus is more marked, but he is able to open his jaws three-fourths of an inch and take large amounts of liquid nourishment. He voided urine several times in bed; it showed no evidences of carboluria. Delirious at frequent intervals during the day.

October 8th.—During the morning he received three grains acidum carbolicum and 20 c.c. anti-tetanic serum. From midnight the delirium and convulsions have grown rapidly worse. Passing urine and fæces in bed. Temperature during past twenty-four hours ranging from 97° to 103°. Pulse rose to 140 during the morning, jaws tightly set, and arching of back firm. Heavy sweating all night and morning. Died suddenly in a convulsion at 2.30 this afternoon.

During the seven days the patient was under treatment he received by the hypodermic method fifty-two grains acidum carbolicum without any evidences of carboluria or other toxic effects of this drug, and 280 c.c. of anti-tetanic serum (Mulford). For the pains accompanying the paroxysms he had during the seven days nine injections of morphia sulphas of one-third grain each. He took an immense quantity of liquid food, and the last few days alcoholic stimulants. Autopsy not allowed.

J. T. L., white, aged thirty-nine years; American; laborer; single; admittep August 17, 1900. Dr. T. H. Weisenberg, Interne. Patient gave following history: August 9th (nine days before) stepped on a nail, the latter penetrating shoe and wounding base of left great toe. Did not feel any inconvenience, and continued at work for a day. The next day (the 10th) foot became very tender and excessively painful. The wounded foot had no treatment but washing in hot water and castile soap and a rind of bacon applied, and rest up to time of admission. On the 14th (five days after accident) first noticed stiffness of jaw and muscles of neck. Throat became sore, with painful deglutition and stiffness of neck, the patient thinking he had "quinsy," to which he is liable. The stiffness of jaws, rigidity of neck muscles, and painful and difficult deglutition continued, growing progressively worse, with shooting pains over the whole body, and especially along the spine and over the diaphragm. Last night opisthotonus developed, with slight spasmodic movements of facial muscles and flexing and uplifting of arm and elbow. History of chancre several years ago. Temperate in habits. Never sick before.

Upon admission, temperature, 99.3°; pulse, 80; respirations, 34. Cannot open jaw more than one-fourth of an inch and cannot protrude tongue. Head retracted and only able to move from side to side one inch on account of rigidity of neck muscles. Chest rigid on account of marked opisthotonus; abdominal muscles prominent and rigid, standing out prominently. Hypersensitive over the entire body, but most marked along the spine. A few slight spasmodic movements were observed during examination. Reflexes absent except knee and Babinski. At base of great left toe there is a small, tender swelling, but no surface abrasion or redness. Urine scanty; specific gravity, 1026, with slight ring of albumin, but no casts.

Treatment.—Free incision over swelling on foot and thoroughly curetted and carbolized, and four grains of acidum carbolicum injected about the wound and dressed with a moist bichloride dressing. During the day there was administered by the hypodermic method, in divided doses, 3,000,000 Mulford's and 1,000,000 Pasteur's anti-tetanic serum.

August 18th.—Grew progressively worse during the night, dying at 7 o'clock this morning. Had three convulsions during the night, at 12, 3 and 7 o'clock, respectively, dying in the last one. The first was of twenty minutes' duration, the second of five minutes, and the last one thirty minutes in duration, and this one so powerful and violent that before the nurses could prevent it the spasm threw the patient out of bed.

He took large amounts of liquid food through a catheter, as well as large amounts of water, as he was very thirsty. From time of admission skin covered with heavy perspiration. Body very rigid immediately after death and extremely cyanosed.

An autopsy, made eight hours after death, showed extreme congestion of all the viscera, and particularly of the brain, spinal cord and the lungs. No lesions found. Cultures made from seat of injury and from spinal canal gave negative results.

THE HOSPITAL TRAINING SCHOOL.

By MARION E. SMITH,
CHIEF NURSE.

The Philadelphia Hospital Training School for Nurses was opened January 5, 1885, with fourteen pupils, under the management of Miss Alice Fisher, as Chief Nurse. It has to-day on duty 109 pupils, and has graduated 563 nurses. Of these the following hold positions in charge of hospitals or training schools, either as superintendents or directresses, while many others hold positions as head nurses of wards, etc. About fifteen per cent. of the graduates have married, and as far as we have been able to ascertain ten only have died. Some have not been heard of for years, though the Alumnæ Association keeps them in touch with the alma mater wonderfully well. Some have gone to other countries as missionaries—to Persia, to Korea, and to China:

Philadelphia Hospital, two; Insane Department, Philadelphia Hospital, as Supervisor, one; Children's Hospital, Philadelphia, one; Jefferson Hospital, Philadelphia, one; Visiting Nurses' Society, Philadelphia, one; Private Hospital, Philadelphia, one; Bryn Mawr Hospital, one; St. Timothy's Hospital, Roxborough, one; City Hospital, Wilkesbarre, one; Women's Homœopathic Hospital, Philadelphia, one; Reading Hospital, Pennsylvania, one; Sunbury Hospital, Pennsylvania, one; Lebanon Hospital, Pennsylvania, one; Drifton County Almshouse, one; Connecticut Training School, New Haven, Conn., two; Deaconess' Home and Hospital, Dayton, Ohio, one; Jewish Hospital, Cincinnati, Ohio, one; City Hospital, Cincinnati, Ohio, one; John Sealy Hospital, Galveston, Texas, one, who withdrew on account of ill health; Danville Hospital, Illinois, one; Altoona Hospital, Pennsylvania, one; Eye, Ear and Throat Infirmary, Washington, D. C., one; Eye, Ear and Throat Infirmary, Boston, Mass., one; Matron College, Racine, Wis., one; Maryland University, one; Hospital, Huntsville, Alabama, one; Hospital, Elmira, N. Y., one; City Hospital, Louisville,

Ky., one; City Hospital, Lynchburg, Va., one; Home and Retreat, Lynchburg, Va., one; Hospital, Mexico, one; Sanitarium, Los Angeles, Cal., one; Women's Hospital, New York, one; Hospital, Norfolk, one; City Hospital, San Francisco, Cal., one; Hospital, Washington, D. C., one; Hospital, Lawrenceville, N. Y., one.

LIST OF THE FORMER RESIDENT PHYSICIANS OF THE PHILADELPHIA HOSPITAL.

By EDWARD R. STONE, M.D., and W. A. N. DORLAND, M.D.

The following list has been prepared by Drs. Stone, Dorland, and C. K. Mills, and has been received with the letter which follows. Any corrections or additions will be gladly received by Dr. Edward R. Stone or by the Editor of these REPORTS.

Philadelphia, October 6, 1900.

DEAR DOCTOR HUGHES:

The List of Blockley ex-Residents published in Volume III, PHILADELPHIA HOSPITAL REPORTS, has been carefully revised and corrected, and the names of those who have served in the hospital since have been added. The number in the third column refers to the year of appointment to the hospital; the fourth column, to the college.

U. of P., University of Pennsylvania; Jeff., Jefferson Medical College; W. M. C., Woman's Medical College of Pennsylvania; Medico-Chi., Medico-Chirurgical College; Univ. City N. Y., University of City of New York; Penna. M. C., Pennsylvania Medical College (extinct).

I should be glad to receive information which would make this list more complete.

Yours sincerely,

EDWARD R. STONE.

ABBOTT, HARVEY N.	2405 N. 19th St., Phila.	79	U. of P.
ABBOTT, W. L.		84	U. of P.
ABEL, FRED. T., dec'd.		76	U. of P.
ALBERTSON, WILLIAM C.	Belvidere, N. J.	86	U. of P.
ALLEMAN, FRANK	Germantown Ave. and Cambria St., Phila.	96	Jeff.
ALEXANDER, CLARA J.	Johnstown, Pa.	89	W. M. C.
ALLEN, HARRISON, dec'd.	1933 Chestnut St., Phila.	61	U. of P.
ALLIS, OSCAR H.	1604 Spruce St., Phila.	66	Jeff.
ALLISON, E. W.	125 S. 18th St., Phila.	80	U. of P.
ALLYN, HERMAN B.	501 S. 42d St., Phila.	85	U. of P.
AMES, ROBERT P.	Springfield, Mass.	81	Jeff.
ANDERSON, GEORGE B., dec.	Latrobe, Pa.	77	Jeff.
ANGENY, GRANVILLE L.	U. S. N.	95	U. of P.
ANGNEY, WILLIAM M.	S. 15th St., Phila.	79	Jeff.
ARMSTRONG, ALEX.	205 S. Broad St., Trenton, N. J.	96	U. of P.
ASHTON, THOMAS G.	1814 S. Rittenhouse Square, Phila.	88	Jeff.
ATLEE, WILLIAM A., JR.		87	U. of P.
AUGÉ, TRUMAN	2802 N. Broad St., Phila.	92	U. of P.
BAKER, P. B. L.	Enfanta, Barbour Co., Ala.	49	U. of P.
BALDWIN, HELEN		95	W. M. C.
BALDY, J. M.	1831 Chestnut St., Phila.	84	U. of P.
BALL, EDWARD S., dec'd.	Zanesville, Ohio	78	
BARDSLEY, GEO. ASHTON	Episcopal Hospital	97	U. of P.
BARKSDALE, R.	Petersburg, Va.	52	U. of P.
BARNES, CHARLES S.	259 S. 15th St., Phila.	98	Jeff.
BARTLES, WILLIAM H.	Flemington, N. J.	65	Jeff.
BARRISTER		43	

BEECHER, A. C. W., dec'd...	Philadelphia	67	Jeff.
BEHREND, MOSES	1331 Franklin St., Phila	99	U. of P.
BELLOWS, HORACE M	Huntingdon Valley, Pa	61	U. of P.
BENTON, CHARLES H., dec'd		59	
BENTON, JOHN H., dec'd	Ogdensburg, N. Y	48	U. of P.
BERENS, BERNARD	2002 Chestnut St., Phila	80	U. of P.
BERENS, JOSEPH, dec'd	New York	75	U. of P.
BERENS, T. PASSMORE		87	U. of P.
BERTOLETTE, D. N.	U. S. N	72	Jeff.
BEYEA, HARRY D	237 S. 13th St., Phila	91	U. of P.
BIRKEY, THOMAS W., dec'd.	245 S. 6th St., Phila	51	U. of P.
BITTING, MIRIAM		89	W. M. C.
BLACK, JOHN J.	New Castle, Del	65	U. of P.
BLACKFORD, BENJAMIN	West'n Lunatic Asylum, Staunton, Va	55	Jeff.
BLISS, A. A	117 S. 20th St., Phila	83	U. of P.
BLOOMFIELD, J. C	Athens, Ga	88	Jeff.
BOARDMAN, CHARLES H	16 Wesley Ave, Evanston, Ill	62	U. of P.
BOENNING, H. C	538 N 6th St., Phila	79	Jeff.
BOSTON, L. NAPOLEON	1531 S. Broad St., Phila	96	Medico-Chi.
BOTSFORD, WILLIAM, dec'd.		68	Jeff.
BOWER, J. L	Reading, Pa	88	Jeff.
BOWMAN, F. S	329 Wharton St., Phila	97	Medico-Chi.
BOYD, JOHN S	19 S. 12th St., Phila	70	U. of P.
BOYER, Z. P	1829 N. Broad St., Phila	81	U. of P.
BRADFIELD, G. M	Philadelphia	91	Jeff.
BRADLEY, ALFRED E	U. S. A	87	Jeff.
BRADLEY, MICHAEL	U. S. N	58	
BRADY, ELLIOT T	1600 Wesley Ave., Evanston, Ill	86	Jeff.
BRAGG, J. C		58	U. of P.
BRAXTON, TOMLIN	Palls, King William Co., Va	54	U. of P.
BRECHEMIN, L	U. S. A	76	U. of P.
BRICK, JOSEPH COLES	1629 Locust St., Phila	95	Jeff.
BRICKER, CHARLES E	2739 Girard Ave., Phila	81	Jeff.
BRISTER, JOHN M	336 N. 5th St., Reading, Pa	98	Medico-Chi.
BROOKE, BENJAMIN	U. S. A	89	U. of P.
BROOKE, HARRIET W., dec'd	Reading, Pa	86	W. M. C.
BROWN, C. H	1824 Diamond St., Phila	78	U. of P.
BROWN, G. S	114½ 21st St., Birmingham, Ala	85	Jeff.
BRUEN, E. T., dec'd	Philadelphia	74	U. of P.
BRUNER, W. E	Cleveland, Ohio	91	U. of P.
BUCK, SAMUEL T		90	
BUCK, W, P	Lansdowne, Pa	69	U. of P.
BUDD, A. V	Lockville, Chatham Co., N. C.	54	U. of P.
BUMSTEAD, CHARLES	Decatur, Ill	98	U. of P.
BUNCE, T. S., dec'd	Philadelphia	82	U. of P.
BURTENSHAW, J. H	381 West End Ave, N. Y	91	U. of P.
BURNETT, J. W., dec'd		62	Jeff.
BURWELL, GEO. N., dec'd	Buffalo, N. Y	43	U. of P.
BUSH, LEWIS P., dec'd	Wilmington, Del	36	U. of P.
BUXTON, JOSEPH T	Newport News, Va	98	U. of P.
CALLAHAN, ANDREW, JR	1639 S. 13th St., Phila	99	Medico-Chi.
CAMPBELL, H. E	100 Sheffield St., Allegheny City, Pa	81	Jeff.
CAMPBELL, H. S	828 N. Broad St., Phila	67	U. of P.
CARPENTER W. H	Salem, N. J	92	U. of P.
CARTER, CHARLES, dec'd	Philadelphia	40	U. of P.
CARTER, W. S	Galveston, Texas	90	U. of P.
CHASE, A. F	3805 Baring St., Phila	74	Jeff.
CHASE, ROBERT H	State Hospital, Norristown, Pa	69	U. of P.
CHRISTIE, S. P.		58	

CLARKE, FLOYD S.	Philadelphia Hospital	00	Medico-Chi.
COHEN, J. SOLIS	1431 Walnut St., Phila.	60	U. of P.
COHEN, MEYER S.	Philadelphia Hospital	00	U. of P.
COLEMAN, J. S., dec'd.	Augusta, Ga.	57	Jeff.
COLEMAN, P. T.	Curdsville, Buckingham Co., Va.	52	
CONE, CLARIBEL	Baltimore, Md.	91	W. M. C.
CORBET, W. B., dec'd.		63	Jeff.
COUSINS, A. S.		58	U. of P.
COWGILL, CLAYTON A.	3609 Baring St., Phila.	46	U. of P.
COYLE, ROBERT	723 S. 16th St., Phila.	85	Jeff.
CRAIG, JAMES A.	Kosaugua, Iowa	95	Jeff.
CRAIG, W. F.	1704 N. 13th St., Phila.	97	
CRAIG, WILLIAM G.	1045 Tioga St., Phila.	92	Jeff.
CRAIG, T. C.	2002 Chestnut St., Phila.	80	U. of P.
CROWELL, ELISHA	21 S. 39th St., Phila.	51	U. of P.
CROWELL, G. M.		82	Jeff.
CRUICE, JOHN M.	225 S. 20th St., Phila.	98	U. of P.
CULLEN, J. S. DORSEY, dec'd	412 E. Grace St., Richmond, Va.	54	
CULPEPPER, W. A., dec'd.	Barbadoes	83	U. of P.
CUMMINGS, J. B.	Forrest City, Ark.	73	Jeff.
CUMMISKEY, JAMES	2107 Arch St., Phila.	56	Jeff.
CUNNINGHAM, EDWARD J.	Philadelphia Hospital	00	U. of P.
CURRY, G. E.	McKeesport, Pa.	92	U. of P.
CURTIN, ROLAND G.	22 S. 18th St., Phila.	66	U. of P.
CURTIS, L. W.	U. S. N.	80	U. of P.
DA COSTA, J. CHALMERS	1629 Locust St., Phila	85	Jeff.
DAGGETT, WILLIAM G.	22 College St., New Haven, Conn.	84	U. of P.
DARBY, J. F., dec'd.		58	U. of P.
DARRACH, GEORGE M.	Cumberland, Monroe Co., Ind.	52	
DASHIEL, T. K.		58	
DAVIS, A. M.	5054 Main St., Germantown, Pa.	92	U. of P.
DAVIS, JAMES A.	527 S. 42d St., Phila.	87	U. of P.
DAVIS, SIDNEY	Milton Pa.	78	U. of P.
DAVIS, T. D.	6018 Penn Ave., Pittsburgh, Pa.	70	Jeff.
DAY, GEORGE E.	Strasburg, Pa.	84	Jeff.
DEASE, STEPHEN S.	India	75	Jeff.
DEAVER, R. W.	6033 Main St., Germantown, Pa.	74	U. of P.
DE BENNEVILLE, J. S.		59	U. of P.
DERCUM, CLARA T.	1115 Fairmount Ave., Phila.	87	W. M. C.
DE SILVER, JOSEPH F.		00	U. of P.
DESSAU, S. HENRY	47 W. 56th St., N. Y.	68	Jeff.
DILLON, J. D., dec'd.	328 S. 5th St., Phila.	79	Jeff.
DILLER, THEO.	200 9th St., Pittsburgh, Pa.	86	U. of P.
DINSMORE, F. M.	Keene, N. H.	94	U. of P.
DIXON, E. D.		96	W. M. C.
DOAN, HENRY H.	53 N. 40th St., Phila.	90	U of P.
DOCK, C.	904 N. Broad St., Phila.	80	Jeff.
DODGE, C. L.		78	Jeff.
DOLAN, W. K.	Scranton, Pa.	80	U. of P.
DONNELLY, M. A., dec'd.	Philadelphia	79	Jeff.
DONOHO, ALBERT P.	Philadelphia Hospital	00	Medico-Chi.
DONOHUE, MICHAEL J.	Waterbury, Conn.	86	U. of P.
DORLAND, W. A. N.	120 S. 17th St., Phila.	86	U. of P.
DORRANCE, HENRY, dec'd.	Philadelphia	47	U. of P.
DRIPPS, J. H.	1812 N. 11th St., Phila.	79	Jeff.
DUER, EDWARD L.	1606 Locust St., Phila.	60	U. of P.
DUHRING, LOUIS A.	1411 Spruce St., Phila.	67	U. of P.
DULLES, CHARLES W.	4101 Walnut St., Phila.	75	U. of P.
DWIGHT, HENRY E.	336 S. 15th St., Phila.	67	U. of P.
DYSON, J. R.	Hazleton, Pa.	97	Medico-Chi.

EBERMAN HENRY F.	Lancaster, Pa.	79	U. of P.
EDGAR, JOHN M.	U. S. N.	80	U. of P.
EDGER, B. J.	U. S. A.	97	U. of P.
EDWARDS, JOSEPH S., dec'd	Atlantic City, N. J.	75	U. of P.
EDWARDS, W. A.	San Diego, Cal.	81	U. of P.
EDWARDS, W. F., dec'd.	Detroit, Mich.	80	U. of P.
ELDER, F. H.	1427 Arch St., Phila.	82	U. of P.
ELMER, HENRY W.	65 W. Commerce St., Bridgeton, N. J.	69	U. of P.
ELMER, WILLIAM, dec'd.	Bridgeton, N. J.	36	U. of P.
ESHELMAN, E. E., dec'd.	Philadelphia	70	Jeff.
ESHNER, A. A.	224 S. 16th St., Phila.	88	Jeff.
ESTERLY, D. E.	Lawrence, Kan.	94	U. of P.
EVANS, E. W.	65 N. 3d St., Easton, Pa.	84	U. of P.
EVERSFIELD, W. C.	Agricultural College, Prince George's Co., Md.	63	U. of P.
FAIRFIELD, J. H.	Great Falls, Mon.	84	U. of P.
FARNHAM, ALICE MAY	Asylum, Hart's Island, N. Y.	84	W. M. C.
FELL, JONATHAN		40	U. of P.
FISHER, WILLIAM E.	Reading, Pa.	99	U. of P.
FITZPATRICK, WM. J.	3648 N. Broad St., Phila.	95	U. of P.
FLICK, L. F.	736 Pine St., Phila.	79	Jeff.
FORD, WILLIAM H., dec'd.	1622 Summer St., Phila.	64	Jeff.
FOX, GEORGE HENRY	18 E. 31st St., N. Y.	69	U. of P.
FRALEY, FREDERICK	Pennsylvania Hospital	99	U. of P.
FRASE		82	
FREE, G. B. M.	Williamson School, Pa.	84	U. of P.
FRENCH, SAMUEL	24 N. Franklin St., Wilkesbarre, Pa.	94	U. of P.
FREUND, H. H.	1310 S. 5th St., Phila.	80	Jeff.
GARRETT, E. F., dec'd.	Philadelphia	77	Jeff.
GAYLORD, H. R.		94	U. of P.
GEISLER, HOWARD D.	202 High St., Germantown	99	U. of P.
GERSON, T. P.	Lansdowne, Pa.	97	U. of P.
GIBB, JOSEPH S.	1804 Chestnut St., Phila.	80	U. of P.
GILBERT, JOHN E., dec'd.	Gettysburg, Pa.	78	U. of P.
GILMORE, A.		91	W. M. C.
GILPIN, SHERMAN	Jefferson Medical College, Phila.	97	Jeff.
GIRVIN, EDWARD R.	Denver, Col.	76	U. of P.
GIRVIN, ROBERT M., dec'd.	3906 Walnut St., Phila.	62	Jeff.
GITHENS, THOMAS S.	835 S. 49th St., Phila.	99	U. of P.
GITHENS, W. H. H.	1337 Pine St., Phila.	66	U. of P.
GODFREY, HENRY G.	Girard College, Phila.	00	Jeff.
GOLDEN, HENRY	710 Pine St., Phila.	97	U. of P.
GOODMAN, H. E., dec'd.	1509 Walnut St., Phila.	59	U. of P.
GORGAS, S. R., dec'd.	Harrisburg, Pa.	75	Jeff.
GOTWALD, D. K.	Springfield, Ohio	82	U. of P.
GRABER, LEON J. K.	Harrisburg, Pa.	79	U. of P.
GRAHAM, A. H.		58	U. of P.
GRAHAM, E. E.	1713 Spruce St., Phila.	87	Jeff.
GRAYSON, CHARLES P.	251 S. 16th St., Phila.	82	U. of P.
GRIFFITH, S. H.	U. S. N., Washington, D. C.	71	U. of P.
GROSS, S. W., dec'd.	Philadelphia	56	Jeff.
GUITÉRAS, DANIEL	329 W. 58th St., N. Y.	74	U. of P.
GUITÉRAS, JOHN	Havana, Cuba	74	U. of P.
GUITÉRAS, G. M.	U. S. Marine Service, 130 S. 39th St., Phila.	85	U. of P.
GUTHRIE, G. W.	47 S. Franklin St., Wilkesbarre, Pa.	73	U. of P.
HACKLEY, C. E.	Unionville, Conn.	60	U. of P.
HAEHNLEN, W. F.	1616 Walnut St., Phila.	82	U. of P.

HAGY, J. A.	Dobbs' Ferry, N. Y.	64	U. of P.
HAINES, JOSIAH		44	Jeff.
HALBERSTADT, GEORGE H.	218 Market St., Pottsville, Pa.	78	U. of P.
HALE, GEORGE	4428 Paul St., Frankford, Phila.	70	U. of P.
HALL, JOHN C., dec'd.	Friend's Asylum, Frankford, Phila.	69	U. of P.
HALL, J. H.	U. S. N	73	U. of P.
HALL, WILLIAM R.		71	U. of P.
HALLOWELL, WILLIAM H.	1338 1st Ave., Minneapolis, Minn.	80	U. of P.
HAMILL, R. H.	Summit, N. J.	79	U. of P.
HAMILL, SAMUEL M.	1822 Spruce St., Phila.	88	U. of P.
HAMMOND, CLARA M.	320 S. 16th St., Phila.	87	W. M. C.
HANCOCK, E. C.		81	U. of P.
HARDY, BENJAMIN F. dec'd.	San Francisco, Cal.	39	U. of P.
HARRIS, CHARLES M. dec'd.	Philadelphia	70	U. of P.
HARRIS, T. J.	106 W. 79th St., N. Y.	89	U. of P.
HARRISON, J. M.	Bryn Mawr, Pa.	79	U. of P.
HAWLEY, B. F.	417 N. 33d St., Phila.	82	Jeff.
HAZLETT, E. E.	Abilene, Dickinson Co., Kan.	80	U. of P.
HEARNE, JAMES C.	Hannibal, Mo.	72	Jeff.
HEATH, WILLIAM H.		78	Jeff.
HELLER, JACOB B.	Easton, Pa.	78	U. of P.
HELM, WILLIAM H., dec'd.	Sing Sing, N. Y.	64	U. of P.
HENDERSON		77	
HENLEY, LEO.	Williamsburg, Va.	47	U. of P.
HENRY, C. P.	U. S. N.	82	U. of P.
HERCHELROTH, J. GRANT	4837 Baltimore Ave. Phila.	94	Medico-Chi.
HETRICK, CAROLINE		95	W. M. C.
HEWITT, GEORGE A.	878 N. 25th St., Phila.	77	Jeff.
HICKMAN, H.	2619 Columbia Ave. Phila.	84	U. of P.
HIGGINBOTHAM, EDW'D G.	239 W. Walnut, Louisville, Ky.	45	U. of P.
HINKLE, WILLIAM H.	1323 N. 13th St., Phila.	92	U. of P.
HITSCHLER, WILLIAM	1208 Spruce St., Phila.	94	U of P.
HITZ, HENRY B.	607 Matthews Building, Milwaukee, Wis.	91	U. of P.
HOFFMAN, JOSEPH E.	126 Diamond St., Phila.	83	U. of P.
HOFFMAN, W. A., dec'd.		68	U. of P.
HOLLAND, DANIEL J., dec'd.	Atchison, Kan.	76	Jeff.
HOLLAND, J. W.		71	
HOLLOWAY, THOMAS B.	5429 Haverford Ave., Phila.	97	U. of P.
HOLMES, E. W.	1930 Chestnut St., Phila.	80	U. of P.
HOONAMAN, G. H.		90	
HORWITZ, L. N., dec'd.		82	Jeff.
HORWITZ, THEO., dec'd.		76	Jeff.
HOUGH, J. STOCKTON, dec'd.	Trenton, N. J.	68	U. of P.
HOUSTOUN, JAS. P. S., dec'd.	98 Harris St., Savannah, Ga.	69	U. of P.
HOUSEKEEPER, F. P.	3508 Baring St., Phila.	74	U. of P.
HOWE, HENRY D.	Hampton, Va.	95	U. of P.
HUG, EDWARD V.	Navarro, Ohio	93	Jeff.
HUGHES, D. E.	Philadelphia Hospital, Phila.	78	Jeff.
HUGHES, F. W.		80	U. of P.
HULL, GEORGE S.	72 W. Main St., Chambersburg, Pa.	77	U. of P.
HUME, J. E.	900 S. 49th St., Phila.	00	U. of P.
HUMPHREY, G. E.	Hazleton, Pa.	90	U. of P.
HUNT, ELIZABETH G.	1602 Arch St., Phila.	89	W. M. C.
HURLOCK, F. J.	2161 N. 29th St., Phila.	82	Jeff.
HUTCHINSON, G. H., dec'd.	Englishtown, N. J.	80	U. of P.
HUTCHINSON, RANDALL	Eckman, W. Va.	87	U. of P.
HUTCHINSON, R. C.	817 Broad St., Trenton, N. J.	80	U. of P.
INGHAM, SAMUEL D.	Trenton, N. J.	99	Medico-Chi.
IRISH, W. B.	6906 Penn Ave., Pittsburgh, Pa.	94	Jeff.

JAMAR, JOHN H.	Elkton, Md.	61	U. of P.
JAMESON, E. W., dec'd.	St. Louis, Mo.	70	U. of P.
JAMESON, WILLIAM B.	761 N. 40th St., Phila.	86	U. of P.
JAMISON, J. ROSS.	Apple Creek, Ohio.	94	
JANNEY, FRANCES S.	Riverton, N. J.	90	W. M. C.
JENKINS, S. R.	Prince Edward's Island.	84	U. of P.
JENKS, WILLIAM F., dec'd.	Philadelphia.	66	U. of P.
JESSOF, S. A. S.	Kittanning, Pa.	80	Jeff.
JEWITT, MARY B.	New York.	96	W. M. C.
JIMINEZ, J. M.	Costa Rica.	69	Jeff.
JIMINEZ, S. M.	73 W. 36th St., N. Y.	79	Jeff.
JOHNSON, N. L.	Williamsport, Pa.	91	U. of P.
JOHNSTON, A. R.	Pierceton, Indiana.	82	Jeff.
JOHNSTON, JOHN.	Dublin, Ireland.	99	U. of P.
KAHN, JOSEPH.	St. Paul, Minn.	89	U. of P.
KARCHER, WILLIAM L.	Freeport, Ill.	00	Medico-Chi.
KEATING, JOHN M., dec'd.	Philadelphia.	74	U. of P.
KEATING, WM. V., dec'd.	Philadelphia.	44	U. of P.
KEEFER, F. R.	U. S. A.	89	U. of P.
KELLER, HARRY M.	Hazleton, Pa.	87	U. of P.
KELLY, E. P. B.		58	Jeff.
KERLIN, E. J.	576 Fullerton Ave., Chicago, Ill.	86	U. of P.
KERR, J. W., dec'd	Allegheny City Pa.	77	Jeff.
KERR, JAMES W.	York, Pa.	39	U. of P.
KETCHAM, S. R	1708 Green St., Phila.	89	U. of P.
KING.		82	
KING, WILLIAM H.	412 S. 15th St., Phila.	94	Jeff.
KIRK, L. H.	Oxford, Pa.	78	U. of P.
KISTLER, CLINTON J.		96	U. of P.
KLEINSTUBER, WILLIAM S.	212 E. 6th St., Wilmington, Del.	93	Jeff.
KLUTTZ, W. C.	Salisbury, N. C	99	U. of P.
KOCH, JAMES L.	Emergency Hosp., Boston, Mass.	96	U. of P.
KOERPER, JOSEPH	Bethayres, Mont. Co., Pa.	62	Phila. Coll.
KNOX, JOHN.	Princeton, Iowa.	77	U. of P.
KOLLOCK, CHARLES W.	Charleston, S. C.	81	U. of P.
KUGLER, G. W.	North Carolina.	85	Jeff.
LAKE, DAVID H.	Kingston, Pa.	85	Jeff.
LANDIS, H. G., dec'd.		70	Jeff.
LANDIS, H. R. M	225 S. 20th St, Phila.	97	Jeff.
LAPIN, F S.		81	
LARGE, OCTAVIUS P	S. E. Cor. 29th and Susq. Av., Phila.	98	Medico-Chi.
LAZARUS, S. D dec'd.	Philadelphia	83	Jeff.
LEE, CHAS. CARROLL, dec'd.	79 Madison Ave., N. Y.	60	U. of P.
LEETE, JAMES M.	2912 Washington Ave., St. Louis.	61	U. of P.
LEIDY, PHILLIP, dec'd.	526 Marshall St., Phila.	59	U. of P.
LEVY, HENRY H.	1407 E. Broad St., Richmond, Va.	71	U. of P.
LEYS, J. L.	U. S. N	91	U. of P.
LICHTY, JOHN A.	4634 5th Ave., Pittsburgh, Pa.	93	
LIGHT, SAMUEL D. W.	Baltimore, Md.	99	U. of P.
LINCOLN CLARENCE W.	5348 Wayne Ave., Phila.	93	U. of P.
LINEAWEAVER, J K.	Columbia, Pa.	61	Jeff.
LINN G. WILDS.		73	U. of P.
LIPPINCOTT FRANKLIN.		40	U. of P.
LITTIG, LAWRENCE W.	Iowa City, Iowa.	84	U. of P.
LITTLE, W T	Canon City, Col.	94	U. of P.
LODER, PERCIVAL E.	517 S. 8th St., Phila.	76	Jeff.
LODGE, JOHN.		59	
LODGE, WILLIAM J.	Baltimore, Md.	59	
LONG, WILLIAM S.	Haddonfield, N. J.	79	U. of P.
LUCAS, EMMA J.		93	W. M. C.

LUDLOW, JOHN L., dec'd	1931 Chestnut St., Phila	41	U. of P.
LUDLOW, R. G.		64	U. of P.
LYMAN, GEORGE H., dec'd.	Boston, Mass.	43	U. of P.
LYON, CHARLES H.	832 N. Broad St., Phila	99	U. of P.
MACCOY, A. W.	1417 Walnut St., Phila	70	U. of P.
MACCRACKEN, G. Y.	612 N. 13th St., Phila	77	U. of P.
MAGOFFIN, M. M.	Mercer, Pa.	62	U. of P.
MALONEY, S. O.		99	U. of P.
MANN, CHARLES W., dec'd.		66	
MARCUS, HERMAN D.	2263 Frankford Ave., Phila	92	Medico-Chi.
MARTIN, CHARLES S.	11 S. 5th St., Allentown, Pa	90	U. of P.
MARTIN, JOSEPH	2009 Columbia Ave., Phila	78	U. of P.
MATLACK, ELWOOD	703½ N. 8th St., Phila	86	U. of P.
MATSON, E. G.	810 Penn Ave., Pittsburgh, Pa	83	U. of P.
MATTHEWS, E. L. B.		91	W. M. C.
MATTHEWS, WILLIAM E.	181 Lincoln St., Johnstown, Pa	87	Jeff.
MAURY, F. F.	Philadelphia	62	Jeff.
MAY, JAMES V.	U. S. A.	97	U. of P.
MCAULEY, JAMES A.	2827 Frankford Ave., Phila	74	U. of P.
MCBRIDE, G. W., dec'd		79	U. of P.
MCCAMY, R. H.	1932 E. Cumberland St., Phila	79	U. of P.
MCCARTHY, DANIEL J.	1342 Pine St., Phila	95	U. of P.
MCCARTY, R. H., dec'd.		79	U. of P.
MCCLEES, WILLIAM D.	Atchison, Kan.	85	U. of P.
MCCLINTOCK, JAMES, dec'd.		58	Jeff.
MCCOY, A.		88	Jeff.
MCCOY, HENRY W.	Golconda, Pope Co., Ill.	64	Jeff.
MCCONKEY, THOMAS G.	San Diego, Cal.	90	U. of P.
MCCLURE, W. W.	21 S. 16th St., Phila	64	Jeff.
MCDONALD, A. A.		74	U. of P.
MCFARLAND, JOSEPH	Philadelphia	89	U. of P.
MCGILL, GEORGE M., dec'd.	Princeton, N. J.	61	U. of P.
MCGLINN, JOHN A.	2204 Fairmount Ave., Phila	99	Medico-Chi.
MCGOWN, D. J.	Los Angeles, Cal.	79	U. of P.
MCGUIGAN, J. J.	Overbrook, Pa.	87	Jeff.
MCKEE, JAMES H.	1519 Poplar St., Phila	92	U. of P.
MCKENYON		74	
MCLAUGHLIN, THOMAS N.	1814 N St., N. W., Washington, D. C.	82	Georget'n Univ.
MCMILLAN, JAMES H., dec'd	St. Louis, Mo.	88	U. of P.
MCPHEETERS, WILLIAM M.	3452 Pine St., St. Louis, Mo.	40	U. of P.
MEANS, J. S.		59	
MEARS, J. EWING	1429 Walnut St., Phila	66	Jeff.
MENAH, W. MCC.	1715 Pine St., Phila	90	U. of P.
MERCUR, JOHN D.	1432 Pine St., Phila	78	Jeff.
MERILLAT, WILLIAM C.		64	U. of P.
MERRITT, VICTOR S.	West Hartford, Conn.	98	Medico-Chi.
MILLIKEN, C. W.	Akron, Ohio	81	U. of P.
MILLIKEN, F. H.	3614 Walnut St., Phila	79	U. of P.
MILLER, MILO G.	40 N. 36th St., Phila	88	U. of P.
MILLER, ROBERT		64	Jeff.
MILLIGAN, JAMES E., dec'd.		72	U. of P.
MITCHELL, JAMES	711 N. 17th St., Phila	83	U. of P.
MOFFITT, WILLIAM J.	1239 N. 2d St., Phila	76	Jeff.
MONTEGUT, SIDNEY		92	
MONTGOMERY, E. E.	1715 Walnut St., Phila	74	Jeff.
MOORE, DUNLOP, JR.	Pittsburgh, Pa.	93	U. of P.
MOORE, EDWARD M.	Rochester, N. Y.	39	U. of P.
MOORE, HENRY B.	Colorado Springs, Col.	86	Jeff.
MOORE, ISAAC H.	Prairie City, Iowa	78	U. of P.
MORGAN, A. C.	3014 Diamond St., Phila	97	Medico-Chi.

MORRIS, S. E.		90	
MOSELY, E. B.	U. S. A.	68	U. of P.
MOSS, WILLIAM	Chestnut Hill, Phila.	54	Jeff.
MOWRY, WILLIAM B.	212 North Ave., Allegheny City, Pa.	76	U. of P.
MOYER, SHERMAN T.	1912 Girard Ave., Phila.	86	U. of P.
MUHLENBERG, F., dec'd.	Lancaster, Pa.	68	U. of P.
MURPHY, JOSEPH A.	328 W. Thompson St., Phila.	98	Medico-Chi.
MURRAY, G. D.	Scranton, Pa.	90	
MURRAY, JAMES M.		76	U. of P.
MURRAY, R. D.	U. S. A.	70	Jeff.
MUSSER, MILTON B., dec'd.	Philadelphia	68	Jeff.
MUSSER, JOHN H.	1927 Chestnut St., Phila.	77	U. of P.
MUTTART, GEORGE W.	505 Communipaw Ave., Jersey City.	91	Univ. City N. Y.
MYERS, SYLVAN	New Orleans, La.	96	U. of P.
NAGLE, FRANK O.		79	U. of P.
NEAD, D. W.	Harrisburg, Pa.	82	U. of P.
NEALE, H. M.	Upper Lehigh Pa.	80	U. of P.
NEARE, C. R.	Orange, N. J.	99	Jeff.
NEWGARDEN, G. J.	U. S. A.	89	Jeff.
NEWTON, R. D.	2705 Oxford St., Phila.	96	Medico-Chi.
NICHOL, WILLIAM L.	Nashville, Tenn.	49	U. of P.
NICHOLS, HENRY D.	Atlantic City, N. J.	98	U. of P.
NICHOLS, W. V.	Oceanside, Cal.	85	U. of P.
NICHOLSON, J. L.	400 Penn St., Camden, N. J.	91	
NICKERSON, L. H. A.	Quincy, Ill.	75	U. of P.
NORRIS, RICHARD F.	Preston Retreat, Phila.	87	U. of P.
NOVAES, F. deP.	Brazil	84	U. of P.
OHNESORG, KARL		95	U. of P.
OLIPHANT, N. B.	110 W. State St., Trenton, N. J.	80	U. of P.
OLIVER, CHARLES A.	1507 Locust St., Phila.	77	U. of P.
O'NEILL, J. WILKS	2110 Spruce St., Phila.	77	U. of P.
O'REILLY, THOMAS B.	2502 Richmond St., Phila.	94	
OWEN, J. J.	411 Pine St., Phila.	80	Jeff.
OWENS, JOHN E.	1806 Michigan Ave., Chicago	62	Jeff.
ORVIS, CHARLES, dec'd.		67	Jeff.
PARISH, WILLIAM H.	1435 Spruce St., Phila.	71	Jeff.
PARKE WILLIAM E.	709 N. 17th St., Phila.	86	U. of P.
PARKHILL, CLAYTON	McPall Building, Denver, Coll.	83	Jeff.
PARRISH, ROBERT C.	Youngstown, O.	98	Medico-Chi.
PARRY, JOHN S., dec'd.	Philadelphia	65	U. of P.
PATTERSON, JOHN P.		71	Jeff.
PECK, ELIZABETH L.	819 N. 40th St., Phila.	85	W. M. C.
PELHAM, J. W.	Asheville, N. C.	88	Jeff.
PEPPER, WILLIAM	1811 Spruce St., Phila.	97	U. of P.
PERKINS, F. M.	1428 Pine St., Phila.	76	U. of P.
PERRY, HEXT M., dec'd.	South Carolina	73	Jeff.
PERRY, JOHN C.		40	U. of P.
PFAHLER, GEORGE E.	Philadelphia Hospital	98	Medico-Chi.
PHILLIPS, WM. L., dec'd.	Pittsburgh, Pa.	76	U. of P.
PHILLIPS, R. J.	4011 Chestnut St., Phila.	83	Jeff.
PHILLRICK, INEZ C.		91	W. M. C.
PICKETT, WILLIAM C.	1508 N. 4th St., Phila.	96	Jeff.
PICOTT, MITCHELL H., dec'd.	Geneva, N. Y.	61	Jeff.
PLUMER, A. J.	Whitman, Neb.	85	U. of P.
POLK, W. ROBESON	414 Nicollet Ave., Minneapolis, Minn.	81	U. of P.
POLLOCK, FLORA		91	W. M. C.
POLTER, THOMAS C.	5920 Green St., Germantown, Pa.	71	U. of P.

PONTIUS, N. D.	70th and Woodland Ave., Phila.	97	Jeff.
PORTER, WILLIAM G.	1118 Spruce St., Phila.	69	U. of P.
PORTER, P. B.	22 W. 31st St., N. Y.	69	U. of P.
POTSDAMER, J. B.	1333 Franklin St., Phila.	80	Jeff.
POTTS, CHARLES S.	1629 Locust St., Phila.	85	U. of P.
PREFONTAINE, L. A.	Springfield, Mass.	92	U. of P.
PRESTON, SAMUEL P.	Lynchburg, Va.	87	Jeff.
PRICE, HELENA J.		86	W. M. C.
PURVES, G. M.	750 S. 10th St., Phila.	96	U. of P.
RABINOVITCH, LOUISA G.		90	W. M. C.
RADEBAUGH, J. M.	Pasadena, Cal.	74	U. of P.
RANDALL, EDWARD, JR.	Galveston, Tex.	83	U. of P.
RANSLEY, ALEXANDER W.	1230 S. 10th St., Phila.	75	U. of P.
RATHBUN, F. D.	New Windsor, Ill.	79	Jeff.
RECTENWALD, JOHN J.	Pittsburgh, Pa.	96	U. of P.
REED, C. H.	121 S. 17th St., Phila.	82	U. of P.
REEDY, WALTER M., dec'd.	Scranton, Pa.	93	
REESER, RICHARD	Municipal Hospital, Phila.	96	Jeff.
REEVE, JOSIAH	Medford, N. J.	64	U. of P
REYNOLDS, CHARLES R.	U. S. A.	99	U. of P.
RIESMAN, DAVID	326 S. 16th St., Phila.	92	U. of P.
REX, OLIVER	Penn Mutual Life Ins. Co., Phila.	67	Jeff.
REYNOLDS, F. P.	U. S. A.	90	U. of P.
RHEIN, JOHN H.	318 S. 15th St., Phila.	90	U. of P.
RICHARDSON, D. D.	Norristown Insane Asylum	58	U. of P.
RICHARDSON, ELLIOTT, dec.	Philadelphia	68	U. of P.
RICHARDSON, GEORGE H.	Missionary, India	91	U. of P.
RICHARDSON, JOHN D.		39	U. of P.
RICIO, SEMFRIO, dec'd.	Cuba	61	
RITZ, CHARLES M.	Lewistown, Pa.	67	U. of P.
ROBERTS, A. S., dec'd.	1901 Walnut St., Phila.	77	U. of P.
ROBERTS, ISAAC E.	1344 N. 13th St., Phila.	67	U. of P.
ROBERTS, THOMAS S.	22 S. 4th St., Minneapolis, Minn.	85	U. of P.
ROBESON, W. F.	Westinghouse Bldg, Pittsburgh, Pa.	85	U. of P.
ROBINSON, EDWIN T.	1326 Pine St., Phila.	00	U. of P.
ROBINSON, ERNEST F.	Lawrence, Kansas	96	U. of P.
ROBINSON, GEORGE S.	House of Correction, Phila.	81	U. of P.
ROBINSON, JOHN M.		91	U. of P.
ROHRER, GEORGE R.	Lancaster, Pa.	81	U. of P.
RONALDSON, WM. R., dec'd.	4017 Locust St., Phila.	74	Jeff.
ROOKER, HERMAN S., dec'd.		85	Jeff.
ROOT, M. P.	India	83	W. M. C.
ROSA, W. V. V.		39	U. of P.
ROSENAU, M. J.	U. S. N.	89	U. of P.
ROTHROCK, J. L.		88	U. of P.
ROUSSEL, A. E.	2112 Pine St., Phila.	82	Jeff.
RUSH, WM. H.	U. S. N.	75	U. of P.
RYNIER, VAN NEST		80	U. of P.
SAGERSON, JOHN L.	Johnstown, Pa.	98	Medico-Chi.
SAILER, J.	248 S. 21st St., Phila.	92	U. of P.
SCHIVELY, GEORGE S.	1503 Gratz St., Phila.	51	Jeff.
SCHNEIDEMAN, T. B.	2725 N. 5th St., Phila.	83	Jeff.
SCHROEDER, HENRY F.	Fennimore, Wis.	99	Medico-Chi.
SEARS, W. H	Huntingdon, Pa.	98	Medico-Chi.
SEDGWICK, W. N.	2012 Tioga St., Phila.	94	Jeff.
SELTZER, C. JAY	1501 Walnut St., Phila.	91	U. of P.
SEYMOUR, A. J.	Fort Washington, Pa.	86	U. of P.
SHARPLESS, CASPER W.		88	U. of P.
SHARPLESS, WILLIAM T.		88	U. of P.
SHEPPARD, J.	36 Commerce St., Bridgeton, N. J.	62	U. of P.

SHERARD, C. C., dec'd		60	U. of P.
SHERK, HENRY H.	Pasadena, Cal	87	Jeff.
SHERMAN, M. H.		91	W. M. C.
SHERRER, FRED. A.		98	Medico-Chi.
SHEW, A. M.	Middleton, Conn	65	Jeff.
SHIELDS, WILLIAM G., JR.	Philadelphia Hospital	00	U. of P.
SHIFFERT, HERBERT O.	U. S. N.	99	Medico-Chi.
SHIMER, WILLIAM S.	713 W. Cumberland St., Phila.	86	U. of P.
SHIMMELL, JAMES S.		81	Jeff.
SHUMWAY, E. A.	2007 Chestnut St., Phila.	94	U. of P.
SMALL, J. F.	135 E. Market St., York, Pa.	89	U. of P.
SMITH, A. S.	3226 Powelton Ave., Phila.	76	Jeff.
SMITH, ALLAN J.	Galveston, Texas	86	U. of P.
SMITH, CHARLES E., JR.	St. Paul, Minn.	65	U. of P.
SMITH, D. K.	Altoona, Pa.	96	U. of P.
SMITH, FISHER, dec'd	U. S. A.	47	U. of P.
SMITH, H. A.	1523 Oxford St., Phila.	75	U. of P.
SMITH, ROBERT K., dec'd		58	
SOMMER, GEORGE N. J.	126 N. Warren St., Trenton, N. J.	94	U. of P.
SOMMERKAMP, R. F.	Philadelphia Hospital	99	U. of P.
SOUTHERN, F. L.	628 N. 16th St., Phila.	90	Jeff.
SPARKS, GEORGE W.	635 Spruce St., Phila.	65	Jeff.
SPEAR, RAYMOND	U. S. N.	95	Jeff.
SPENCE, J. E.		70	Jeff.
SPENCER, THOMAS R.		40	U. of P.
STACKHOUSE, C. P.	Wilkesbarre, Pa.	98	Medico-Chi.
STAHL, B. F.	1502 Arch St., Phila.	87	U. of P.
STAMM, E. P., dec'd		81	U. of P.
STEELE, J. DUTTON	N. E. cor 40th and Locust Sts., Phila.	93	U. of P.
STEHMAN, H. B.	Presbyterian Hospital, Chicago	78	Jeff.
STENGEL, ALFRED	1811 Spruce St., Phila.	89	U. of P.
STELWAGON, H. W.	223 S. 17th St., Phila.	75	U. of P.
STETSON, J. B.	1327 Spruce St., Phila.	97	U. of P.
STEVENS, A. A.	318 S. 15th St., Phila.	87	U. of P.
STEWART, A. H.	250 N. 12th St., Phila.	92	Jeff.
STEWART, W. H., dec'd	2620 N. 11th St., Phila.	82	U. of P.
STEWART, WALTER M., dec.		65	U. of P.
STILLÉ, ALFRED, dec'd	3900 Spruce St., Phila.	36	U. of P.
STIVERS, CHARLES G.	Tonawanda, N. Y.	92	U. of P.
STONE, EDWARD R.	1701 Master St., Phila.	72	Jeff.
STRYKER, S. S.	3833 Walnut St., Phila.	66	U. of P.
SUTTON, R. S.	419 Penn Ave., Pittsburgh. Pa.	65	U. of P.
TABB, JOHN B.		43	Jeff.
TAGGART, THOMAS D.	1825 Fairmount Ave., Phila.	96	Medico-Chi.
TAGGART, WM. H., dec'd		52	U. of P.
TALLY, FRANK W.	1234 Spruce St., Phila.	87	U. of P.
TALLY, JAMES E.	55th and Lancaster Ave., Phila.	92	U. of P.
TAYLOR, G. B.	Barclay, Pa.	85	U. of P.
TAYLOR, J. L.		58	U. of P.
TAYLOR, ROBERT A.	Duluth, Minn.	86	Jeff.
TAYLOR. SARAH M.		90	W. M. C.
THOMAS, ADA R.	Washington, D. C.	93	W. M. C.
THOMPSON, dec'd	Pittsburgh, Pa.	78	
TOPPING, G. G.	Denver, Col.	73	Jeff.
TRUBY, WILLARD F.	U. S. A.	98	U. of P.
TUCKER, HENRY	19 S. 21st St., Phila.	94	Jeff.
TUTEUR, EDWIN B.	La Crosse, Wis.	90	Jeff.
TUTTLE, JAMES P.	42 W. 50th St., N. Y.	81	U. of P.
UPSHUR, GEORGE L.		43	U. of P.

VANDERVOORT, C. A.	3253 N. 15th St., Phila.	94	
VAN EPPS, CLARENCE		99	U. of P.
VAN GASKEN, F. C.	1132 Spruce St., Phila.	91	W. M. C.
VAN HARLINGEN, A.	118 S. 17th., Phila.	68	U. of P.
VANNEMAN, W. S.	Persia	89	U. of P.
VAN VALZAH, W. W.	10 E. 43d St., N. Y.	76	Jeff.
VOGLER, GEORGE W, dec'd.	565 N. 5th St., Phila.	77	U. of P.
VOORHEES, N. W.	Elizabeth, N. J.	83	U. of P.
VOORHEES, SHEPARD		89	U. of P.
WALKER, JAMES B.	1617 Green St., Phila.	72	U. of P.
WALKER, JOHN S.		71	U. of P.
WALKER, THOMAS L., dec'd.	Lynchburg, Va.	39	U. of P.
WALLACE, JOSEPH D.		95	Medico-Chi
WALLACE, WILLIAM H., JR.		64	U. of P.
WALTERS, CHARLES, dec'd.		88	U. of P.
WAPLES, M. H.	Dubuque, Iowa	65	Jeff.
WARE, J. D.	114 N. Delaware Ave., Phila.	76	U. of P.
WARING, JAMES J., dec'd.	Savannah, Ga.	52	U. of P.
WEHNER, WILLIAM H.	329 Wistar St., Germantown, Phila.	87	Jeff.
WEIDMAN, W. M.	214 S. 5th St., Reading, Pa.	60	U. of P.
WEIGHTMAN, JOHN F., dec.	Philadelphia	66	U. of P.
WEISENBURG, THEO. H.	U. S. A.	99	U. of P.
WELLS, GEORGE M.	Wayne Pa.	85	U. of P.
WELLS, P. F.	4023 Brown St., Phila.	82	U. of P.
WELSH, JOHN C.	Belleview, Pa.	93	
WERKEL		77	
WESTON, GEORGE D.	98 Main St., Springfield, Mass.	87	U. of P.
WETHERILL, H. E.	3734 Walnut St., Phila.	95	U. of P.
WETHERILL, H. M.	1420 Chestnut St., Phila.	78	U. of P.
WHELAN, ALFRED		74	U. of P.
WHITE, COURTLAND Y.	334 S. 16th St., Phila.	95	U. of P.
WHITE, J. WILLIAM	1810 S. Rittenhouse Square, Phila.	73	U. of P.
WHITEHEAD, P. F.		59	Jeff.
WILLARD, DE FOREST	1818 Chestnut St., Phila.	67	U. of P.
WILLETTS, E. MILES	104 Adams St., Memphis, Tenn.	55	Jeff.
WILLIAMS, AUGUSTA G.	Brookline, Mass.	95	W. M. C.
WILLIAMS, E. NEWLIN	New Hope, Pa.	99	U. of P.
WILLITTS, C. H.	2015 Chestnut St., Phila.	79	U. of P.
WILLS, W. L.		82	U. of P.
WILSON, F. G.		91	U. of P.
WILSON, JAMES F., dec'd.	Philadelphia	65	U. of P.
WILSON, JOHN J.		43	U. of P.
WILSON, W. R.	267 S. 21st St., Phila.	87	Jeff.
WOLLERTON, S. H.	Oxmoor, Ala.	80	U. of P.
WOOD, B. S.		58	
WOOD, GEORGE, dec'd.	U. S. A.	47	U. of P.
WOOD, H. C.	1925 Chestnut St., Phila.	62	U. of P.
WOODBURY, FRANK T.	U. S. A.	99	Medico-Chi.
WOODHOUSE, SAMUEL W.	1306 Pine St., Phila.	47	U. of P.
WOODRUFF, CHARLES E.	U. S. A.	86	Jeff.
WOODS, C. H.	San Francisco, Cal.	97	U. of P.
WOODS, D. F.	1501 Spruce St., Phila.	64	U. of P.
WOODVILLE, J. L.	Sweet Springs, Monroe Co., Va.	44	U. of P.
YEAGER, GEORGE C.		99	Jeff.
YENNEY, ROBERT C.		93	U. of P.
YOUNG, JAMES K.	222 S. 16th St., Phila.	84	U. of P.
YOUNG, W. W.	Nanticoke, Pa.	94	U. of P.
ZACHERLE, O. F.	3012 Stiles St., Phila.	87	Jeff.
ZERFING, CHARLES E.	Lead, S. Dakota	95	U. of P.
ZIMMER, JOHN	46 Rhine St., Rochester, N. Y.	93	U. of P.

CHRONOLOGICAL LIST OF MEMBERS

OF THE

Medical Boards of the Philadelphia Hospital

FROM 1768 to 1900.

By CHARLES K. MILLS, M.D.

[This list probably contains many omissions and not a few mistakes, as many difficulties have attended its preparation, in which have been consulted Dr. Agnew's "Medical History of the Philadelphia Almshouse," Thatcher's "Medical Biography," Ruschenberger's "History of the College of Physicians," the Catalogues of the Alumni of the Medical Department of the University of Pennsylvania and of the Jefferson Medical College, the Annual Statements of the Boards of Guardians of the Poor and of the Board of Charities and Correction, and the written minutes of the Governing Boards since 1859. Members of the Medical Board have also been personally consulted in efforts at verification. I shall be glad to receive any corrections or additions from any one who may examine the table.—C. K. M.]

MEDICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Cadwalader Evans	1768		
Thomas Bond	1768	1779	
Adam Kuhn	1774	1776	
Benjamin Rush	1774	1777	
Samuel Duffield	1774	1801	
Girardus Clarkson	1774	1777	
Girardus Clarkson	1788	1790	
Thomas Parke	1774	1779	
George Glentworth	1779	1781	
D. Jackson	1779	1781	
James Hutchison	1780	1781	Out-door physician.
—— Wilson	1780	1781	Out-door physician.
Caspar Wistar	1788	1790	
J. R. Rodgers	1788	1789	
Michael Leib	1788	1790	
John Morris	1788	1789	
Samuel P. Griffits	1788	1789	
N. B. Waters	1789	1790	
William Shippen	1789	1790	
—— Cumming	1795		

MEDICAL STAFF.—*Continued.*

Name.	Service began.	Service ended.	Remarks.
—— Pleasants		1797	Date of appointment not known.
Samuel Clements, Jr.	1796	1797	
William Boyce	1796	1801	
Samuel Cooper	1796	1796	
John Church	1797	1805	
Thomas C. James	1797	1811	Transferred to Obstetrical Staff.
John Proudfit	1801	1804	
Philip S. Physick	1801	1805	
Charles Caldwell	1801	1804	
Elijah Griffiths	1801	1810	
Benjamin L. Barton	1804	1805	
Samuel Stewart	1804	1810	
John Rush	1804		Elected, but declined to serve. Dr. Agnew mentions a Dr. Rush as resigning in 1821.
James Reynolds	1804	1807	
James Hutchinson	1805	1805	Served three months.
Isaac Cathrall	1805	1811	Transferred to Surgical Staff.
Peter Muller	1805	1811	Transferred to Surgical Staff.
John Syng Dorsey	1805	1811	
John Syng Dorsey	1814	1815	
Nathaniel Chapman	1807	1815	
Nathaniel Chapman	1822	1832	
Joseph Parrish	1807	1811	Transferred to Surgical Staff.
Samuel Stewart	1810	1822	
Joseph Klapp	1810		
Joseph Klapp	1815	1822	
Thomas Hewson	1811	1822	
Joseph Hartshorne	1818	1820	
Samuel Calhoun	1821	1822	
William P. C. Barton	1821	1822	
William E. Horner	1822	1833	Transferred to Surgical Staff.
Samuel Jackson	1822	1845	
John K. Mitchell	1822	1827	
Richard Harlan	1822	1822	Transferred to Surgical Staff.
Hugh L. Hodge	1822	1835	
Samuel George Morton	1827	1835	
Jacob Randolph	1832	1837	
William H. Gerhard	1835	1845	
Joseph Pancoast	1835	1837	Transferred to Surgical Staff.
William Ashmead	1835	1838	
William Ashmead	1841	1845	
N. Stuardson	1837	1838	
Robley Dunglison	1838	1845	
Edward Peace	1838	1841	
Meredith Clymer	1843	1845	

SURGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
J. Cathrall	1811	1822	Transferred from Medical Staff.
Peter Miller	1811	1822	Transferred from Medical Staff.
Joseph Parrish	1811	1821	Transferred from Medical Staff.
John Rhea Barton	1820	1822	
William Gibson	1821	1822	
J. V. O. Lawrence	1822	1822	
Richard Harlan	1822	1838	Transferred from Medical Staff.
William E. Horner	1832	1835	Transferred from Medical Staff.
Joseph Pancoast	1837	1845	Transferred from Medical Staff.
Charles Bell Gibson	1838	1840	

OBSTETRICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Thomas C. James	1811	1821	Transferred from Medical Staff.
John Moore	1818	1821	
Henry Neill	1821	1835	
Nathan Shoemaker	1821	1827	
Charles Lukens	1827	1827	
B. Ellis	1827	1831	
F. S. Beattie	1831	1837	
Charles Wistar Pennock	1835	1845	
William D. Brinkle	1837	1839	
Charles Bell Gibson	1838	1840	
Robert M. Huston	1839	1845	
James McClintock	1840	1841	
William H. Gillingham	1841	1845	

Administration under Chief Resident Officer.

PHYSICIAN-IN-CHIEF.

Name.	Service began.	Service ended.	Remarks.
H. S. Patterson	1845	1845	

CONSULTANTS.

Name.	Service began.	Service ended.	Remarks.
William Byrd Page	1845		Consulting surgeon.
Meredith Clymer	1845		Consulting physician.
N. D. Benedict	1845	1845	Consulting accoucheur. Office abolished November 9, 1845.

MEDICAL STAFF.—*Continued.*

Name.	Service began.	Service ended.	Remarks.
—— Pleasants...		1797	Date of appointment not known.
Samuel Clements, Jr.....	1796	1797	
William Boyce.....	1796	1801	
Samuel Cooper.....	1796	1796	
John Church.	1797	1805	
Thomas C. James.....	1797	1811	Transferred to Obstetrical Staff.
John Proudfit.	1801	1804	
Philip S. Physick.....	1801	1805	
Charles Caldwell.....	1801	1804	
Elijah Griffiths.	1801	1810	
Benjamin L. Barton.....	1804	1805	
Samuel Stewart.....	1804	1810	
John Rush.	1804		Elected, but declined to serve. I Agnew mentions a Dr. Rush resigning in 1821.
James Reynolds.....	1804	1807	
James Hutchinson.....	1805	1805	Served three months.
Isaac Cathrall.....	1805	1811	Transferred to Surgical Staff.
Peter Muller...	1805	1811	Transferred to Surgical Staff.
John Syng Dorsey.....	1805	1811	
John Syng Dorsey..	1814	1815	
Nathaniel Chapman.....	1807	1815	
Nathaniel Chapman.....	1822	1832	
Joseph Parrish.	1807	1811	Transferred to Surgical Staff.
Samuel Stewart.....	1810	1822	
Joseph Klapp.....	1810		
Joseph Klapp	1815	1822	
Thomas Hewson.	1811	1822	
Joseph Hartshorne.	1818	1820	
Samuel Calhoun.	1821	1822	
William P. C. Barton.....	1821	1822	
William E. Horner.....	1822	1833	Transferred to Surgical Staff.
Samuel Jackson.....	1822	1845	
John K. Mitchell.....	1822	1827	
Richard Harlan.....	1822	1822	Transferred to Surgical Staff.
Hugh L. Hodge.....	1822	1835	
Samuel George Morton....	1827	1835	
Jacob Randolph....	1832	1837	
William H. Gerhard.	1835	1845	
Joseph Pancoast..	1835	1837	Transferred to Surgical Staff.
William Ashmead.....	1835	1838	
William Ashmead.....	1841	1845	
N Stuardson	1837	1838	
Robley Dunglison.....	1838	1845	
Edward Peace.....	1838	1841	
Meredith Clymer.....	1843	1845	

SURGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
J. Cathrall	1811	1822	Transferred from Medical Staff.
Peter Miller	1811	1822	Transferred from Medical Staff.
Joseph Parrish	1811	1821	Transferred from Medical Staff.
John Rhea Barton	1820	1822	
William Gibson	1821	1822	
J. V. O. Lawrence	1822	1822	
Richard Harlan	1822	1838	Transferred from Medical Staff.
William E. Horner	1832	1835	Transferred from Medical Staff.
Joseph Pancoast	1837	1845	Transferred from Medical Staff.
Charles Bell Gibson	1838	1840	

OBSTETRICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Thomas C. James	1811	1821	Transferred from Medical Staff.
John Moore	1818	1821	
Henry Neill	1821	1835	
Nathan Shoemaker	1821	1827	
Charles Lukens	1827	1827	
B. Ellis	1827	1831	
F. S. Beattie	1831	1837	
Charles Wistar Pennock	1835	1845	
William D. Brinkle	1837	1839	
Charles Bell Gibson	1838	1840	
Robert M. Huston	1839	1845	
James McClintock	1840	1841	
William H. Gillingham	1841	1845	

Administration under Chief Resident Officer.

[illegible]

[illegible]	[illegible]	[illegible]	[illegible]
H. S. [illegible]	[illegible]	[illegible]	

[illegible]

[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]		[illegible]
[illegible]	[illegible]		[illegible]
[illegible]	[illegible]	[illegible]	[illegible]

PHYSICIANS-IN-CHIEF.

Name.	Service began.	Service ended.	Remarks.
N. D. Benedict	1845	1850	
—— Haines	1850	1853	
J. D. Stewart	1853	1854	
R. T. Coleman	1854	1854	
Archibald B. Campbell	1854	1854	

Administration by Residents-in-Chief and Board of Clinical Lecturers.

RESIDENTS-IN-CHIEF.

Name.	Service began.	Service ended.	Remarks.
Archibald B. Campbell	1854	1855	
Robert K. Smith	1855	1856	
Archibald B. Campbell	1856	1857	
James McClintock	1857	1858	All the visiting physicians resigned soon after the election of Dr. McClintock, as clinical instruction was abandoned.
Robert K. Smith	1858	1859	

LECTURERS ON CLINICAL MEDICINE.

Name.	Service began.	Service ended.	Remarks.
J. L. Ludlow	1854	1857	
Robert Coleman	1854	1854	Did not accept election.
Gaspas Morris	1854	1855	Transferred to Obstetrical Department.
Joseph Carson	1855	1857	
Joseph Carson	1858	1859	
J. B. Biddle	1855	1857	
J. B. Biddle	1858	1859	
J. Aitken Meigs	1858	1859	
Samuel Dickson	1858	1858	Declined on account of ill health.
J. M. Da Costa	1858	1859	

LECTURERS ON CLINICAL SURGERY.

Name.	Service began.	Service ended.	Remarks.
Henry H. Smith	1854	1857	
D. H. Agnew	1854	1857	
D. H. Agnew	1858	1859	
John Neill	1855	1857	
R. P. Thomas	1855	1857	
W. S. Halsey	1858	1859	
Richard J. Levis	1858	1859	

LECTURERS ON OBSTETRICS AND DISEASES OF WOMEN AND CHILDREN.

Name.	Service began.	Service ended.	Remarks.
R. A. F. Penrose	1854	1857	
R. A. F. Penrose	1858	1859	
Wilson Jewell	1855	1857	
Caspar Morris	1855	1857	Transferred from Medical Department.
E. McClellan	1858	1859	

MEDICAL STAFF.[1]

Name.	Service began.	Service ended.	Remarks.
J. L. Ludlow	1859	1885	
William F. Maybury	1859	1861	
Charles P. Tutt	1859	1866	
Robert Sucket	1859	1859	
J. M. Da Costa	1859	1865	
O. A. Judson	1861	1863	
George J. Ziegler	1863	1867	Transferred from Medical Staff.
Alfred Stillé	1865	1872	
J. S. De Benneville	1866	1866	
Edward Rhoads	1866	1870	
William Pepper	1867	1884	
H. C. Wood	1870	1883	Transferred to Neurological Staff.
James Tyson	1872	1889	
James Tyson	1893		Still serving.
John M. Keating	1875	1877	
John M. Keating	1878	1880	Transferred to Obstetrical Staff.
Edward T. Bruen	1875	1889	
James C. Wilson	1875	1889	
John Guitéras	1875	1880	
Roland G. Curtin	1880		Still serving.
S. J. McFerran	1880	1884	
J. T. Eskridge	1882		Elected, but did not serve.
W. G. McConnell	1882		Elected, but did not serve.
Joseph F. Neff	1884	1887	
John H. Musser	1885		Still serving.
William Osler	1885	1889	
F. P. Henry	1888		Still serving.
J. M. Anders	1889	1896	
William E. Hughes	1889		Still serving.
S. Solis-Cohen	1889		Still serving.
Eugene L. Vansant	1889	1896	
F. A. Packard	1892	1900	
Judson Daland	1892	1895	

[1] At this time the administration by a visiting medical board was resumed.

MEDICAL STAFF.—*Continued.*

Name.	Service began.	Service ended.	Remarks.
Samuel Wolfe	1892	1900	
Julius Salinger	1892		Still serving.
H. A. Hare	1894		Elected, but did not serve.
Thomas G. Ashton	1894		Still serving.
A. A. Eshner	1896		Still serving.
Alfred Stengel	1896		Still serving.
Herman B. Allyn	1900		Still serving.
David Reisman	1900		Still serving.

SURGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Samuel D. Gross	1859	1865	
D. Hayes Agnew	1859	1865	
R. J. Levis	1859	1870	
R. J. Levis	1882		Elected, but did not serve.
Edward L. Duer	1862	1863	Transferred to Obstetrical Staff.
R. S. Kenderdine	1859	1865	
J. W. Lodge	1864	1868	
W. H. Pancoast	1865	1885	
F. F. Maury	1865	1878	Transferred from Obstetrical Staff.
John H. Brinton	1866	1882	
Harrison Allen	1870	1878	
Samuel W. Gross	1874	1882	
N. L. Hatfield	1875	1884	
J. William White	1875	1889	
J. William White	1892	1898	
William G. Porter	1875	1895	
A. A. McDonald	1878	1881	
W. S. Janney	1877	1890	
George McClellen	1880	1890	
A. S. Roberts	1881	1887	
W. Joseph Hearn	1882		Still serving.
C. H. Thomas	1882	1884	
A. W. Ransley	1885	1892	
Lewis W. Steinback	1885		Still serving.
John Blair Deaver	1887	1899	
Edward Martin	1888	1889	
Edward Martin	1892		Still serving.
Orville Horwitz	1889		Still serving.
Ernest Laplace	1889		Still serving.
James M. Barton	1890	1899	
J. Chalmers Da Costa	1895		Still serving.
Alfred C. Wood	1895		Still serving.
Charles H. Frazier	1898		Still serving.
R. A. F. Penrose	1859	1867	

OBSTETRICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
John Wiltbank	1859	1859	
William D. Stroud	1859	1863	
Lewis Harlow	1859	1862	
George J. Ziegler	1859	1863	Transferred to Medical Staff.
A. H. Smith	1862	1864	
E. Schofield	1863	1870	
F. F. Maury	1864	1865	Transferred to Surgical Staff.
Edward L. Duer	1863	1883	Transferred from Surgical Staff.
R. M. Girvin	1865	1876	
J. S. Parry	1867	1876	
George Pepper	1870	1872	
J. V. Ingham	1872	1874	
W. A. Warder	1874	1881	
J. R. Burden, Jr.	1874	1876	
E. E. Montgomery	1877	1884	
E. E. Montgomery	1886	1894	
James B. Walker	1876	1880	Transferred to Medical Staff.
S. S. Stryker	1876	1889	
G. W. Linn	1876	1882	
M. B. Musser	1877	1887	
W. H. Parish	1876	1889	
John M. Keating	1880	1890	Transferred from Medical Staff.
Clara Marshall	1882	1895	
E. P. Bernardy	1882	1884	
Hannah P. Croasdale	1882		Elected, but did not serve.
Theophilus Parvin	1884	1892	
Donnell Hughes	1884	1884	
Eliott Richardson	1886	1886	
Barton C. Hirst	1887		Still serving.
Edward P. Davis	1889		Still serving.
William Easterly Ashton	1889	1894	
Robert H. Hamill	1890	1896	
George I. McKelway	1890		Still serving.
J. W. West	1892	1894	
R. C. Norris	1894		Still serving.
J. M. Fisher	1894		Still serving.
W. Frank Haehnlen	1895		Still serving.
Elizabeth L. Peck	1895		Still serving.
John B. Shober	1896		Still serving.

NEUROLOGICAL STAFF.

Name.	Service began.	Service ended.	Remarks.
Charles K. Mills	1877		Still serving.
H. C. Wood	1883	1887	
H. C. Wood	1887	1888	
Robert Bartholow	1887	1888	

NEUROLOGICAL STAFF.—*Continued.*

Name.	Service began.	Service ended.	Remarks.
Francis X. Dercum	1887		Still serving.
James Hendrie Lloyd	1888	1889	
James Hendrie Lloyd	1890	1900	
Wharton Sinkler	1888	1896	
C. H. Bradfute	1889	1890	
Charles W. Burr	1896		Still serving.
F. Savery Pearce	1901		Still serving.
William C. Spiller	1901		Still serving.
Charles S. Potts	1901		Still serving.

OPHTHALMOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
E. O. Shakespeare	1877	1889	
G. E. de Schweinitz	1887		Still serving.
Charles H. Thomas	1888	1888	Acting ophthalmologist.
George M. Gould	1889	1894	
Charles A. Oliver	1894		Still serving.
Howard F. Hansell	1900		Still serving.
John W. Crosky	1901		Still serving.

DERMATOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
F. F. Maury	1870	1870	Acting dermatologist.
Louis Duhring	1870	1877	
Louis Duhring	1877	1889	Acting dermatologist.
Henry W. Stelwagon	1887		Still serving.
J. A. Cantrell	1889	1900	
Milton B. Hartzell	1900		Still serving.
E. S. Gans	1900		Still serving.

LARYNGOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
C. Jay Seltzer	1890	1900	
George M. Marshall	1890		Still serving.
Charles P. Grayson	1901		Still serving.
E. B. Gleason	1901		Still serving.

PATHOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
James Tyson	1871	1875	
R. M. Bertolet	1872		
Joseph Berens	1876	1879	
E. O. Shakespeare	1882	1889	
Henry F. Formad	1887	1892	
John Guitéras	1892	1899	
W. M. L. Coplin	1892	1895	

PATHOLOGISTS.—*Continued.*

Name.	Service began.	Service ended.	Remarks.
H. W. Cattell.	1898	1900	
E. B. Sangree	1895	1895	
W. M. L. Coplin	1896		Still serving.
Joseph McFarland	1900		Still serving.
Simon Flexner	1900		Still serving.

PEDIATRISTS.

Name.	Service began.	Service ended.	Remarks.
W. C. Hollopeter	1901		Still serving.
Edward E. Graham	1901		Still serving.
J. P. Crozer Griffith	1901		Still serving.
J. Madison Taylor	1901		Still serving.

BACTERIOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
E. O. Shakespeare	1889	1894	
A. Ghriskey	1894	1896	
L. N. Boston	1898		Still serving.

ASSISTANT PATHOLOGISTS.

Name.	Service began.	Service ended.	Remarks.
L. L. Hatch	1889	1891	
H. W. Cattell	1889	1895	
William B. Jameson	1892	1900	
Ernest B. Sangree	1892	1895	
David Bevan	1892	1895	

CURATORS.

Name.	Service began.	Service ended.	Remarks.
D. Hayes Agnew	1860	1867	
William Pepper	1867	1871	
R. M. Bertolet	1871	1872	
R. M. Bertolet	1875	1876	
James Tyson	1872	1875	
Joseph Berens	1876	1879	
E. O. Shakespeare	1880	1882	

MICROSCOPISTS.

Name.	Service began.	Service ended.	Remarks.
James Tyson	1866	1872	
R. M. Bertolet	1872	1875	
Thomas B. Reed	1875	1876	
H. F. Formad	1880	1892	

PHYSICIANS TO THE INSANE DEPARTMENT.

Name.	Service began.	Service ended.	Remarks.
L. Henley	1849	1852	Assistant physician in charge.
J. H. Benton	1852	1852	Assistant physician in charge.
L. Henley	1852	1854	From 1854 to 1859 no one regularly in charge.
Samuel W. Butler	1859	1866	
D. D. Richardson	1866	1880	
D. D. Richardson	1881	1885	
A. A. McDonald	1880	1881	
Philip Leidy	1885	1887	
William H. Wallace	1887	1887	
George M. Wells	1887	1890	
Daniel R. Hughes	1890		Still serving.

CONSULTING PHYSICIANS TO THE INSANE DEPARTMENT.

Name.	Service began.	Service ended.	Remarks.
S. Weir Mitchell	1884	1886	
Horatio C. Wood	1884	1885	
Charles K. Mills	1884	1887	
Charles K. Mills	1890		Still serving.
Andrew Nebinger	1885	1886	
James A. Simpson	1886	1887	
Philip Leidy	1886	1887	
F. X. Dercum	1890		Still serving.
Wharton Sinkler	1890	1896	
James Hendrie Lloyd	1890	1900	
Charles W. Burr	1896		Still serving.
F. Savery Pearce	1901		Still serving.
William C. Spiller	1901		Still serving.
Charles S. Potts	1901		Still serving.

Registrars.

MEDICAL.

Name.	Service began.	Service ended.	Remarks.
W. A. Edwards	1885	1886	
C. J. Seltzer	1886	1890	Elected laryngologist.
F. A. Packard	1890	1892	Transferred to Medical Staff.
Alfred Stengel	1892	1895	Transferred to Medical Staff.
H. Toulmin	1895	1897	
H. B. Allyn	1895	1900	Transferred to Medical Staff.
B. F. Stahl	1900		Still serving.
Joseph Sailer	1900		Still serving.

SURGICAL.

Name.	Service began.	Service ended.	Remarks.
Edward Martin	1885	1888	Transferred to Surgical Staff.
C. B. Penrose	1888	1892	
J. C. Da Costa	1892	1895	Transferred to Surgical Staff.
John H. Gibbon	1895	1900	
R. B. Newton	1900		Still serving.

OBSTETRICAL.

Name.	Service began.	Service ended.	Remarks.
H. A. Pardee	1885	1887	
Edward P. Davis	1887	1888	
R. H. Hamill	1888	1890	
R. C. Norris	1890	1894	Transferred to Obstetrical Staff.
W. A. N. Dorland	1898		Still serving.

NERVOUS.

Name.	Service began.	Service ended.	Remarks.
Guy Hinsdale	1885	1892	
Augustus A. Eshner	1891	1896	Transferred to Medical Staff.
B. F. Stahl	1896	1900	Appointed Medical Registrar.
W. C. Pickett	1900		Still serving.

PEDIATRICS.

Name.	Service began.	Service ended.	Remarks.
James H. McKee	1901		Still serving.

ANÆSTHETIZER.

Name.	Service began.	Service ended.	Remarks.
Charles Lester Leonard	1898		Still serving.

DENTAL SURGEONS.

Name.	Service began.	Service ended.	Remarks.
R. H. Nones	1901		Still serving.
M. H. Cryer	1901		Still serving.
I. N. Broomell	1901		Still serving.
Thos. C. Stellwagon, Jr.	1901		Still serving.

MEMBERS OF THE MEDICAL BOARD.

WITH ADDRESSES, PLACE AND TIME OF GRADUATION, DATE OF APPOINTMENT TO THE PHILADELPHIA HOSPITAL, AND POSITIONS HELD IN OTHER INSTITUTIONS.

In the main this list represents the order of seniority of the different members of the medical board; but in a few instances it does not, as some of the present members are serving for a second period. Some also have been elected during the year, or even at the same meeting of the governing board, and practically the latter do not differ in seniority.

JAMES TYSON, M.D., 1506 Spruce Street. Graduate of University of Pennsylvania, 1863. Appointed, 1872; served until 1889; reappointed, 1893. Professor of Clinical Medicine in the University of Pennsylvania, and Physician to the University Hospital.

CHARLES K. MILLS, M.D., 1909 Chestnut Street. Graduate of University of Pennsylvania, 1869. Appointed, 1877. Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania, and Dean of the Faculty of the Auxiliary Department of Medicine in the University; Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania; Consulting Physician to St. Clement's Hospital for Epileptics; Consultant to St. Joseph's Hospital, to the West Philadelphia Hospital for Women, to the New Jersey Training School for Feeble-Minded Children, and to the Training School for Feeble-Minded Children at Elwyn.

ROLAND G. CURTIN, M.D., 22 South Eighteenth Street. Graduate of University of Pennsylvania, 1866. Appointed, 1880. Consulting Physician to the Rush Hospital for Consumptives, to St. Timothy's Hospital, and to the Midnight Mission; Visiting Physician to the Presbyterian Hospital; Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the Hospital of the University of Pennsylvania; President of the American Climatological Society.

W. JOSEPH HEARN, M.D., 1130 Walnut Street. Graduate of Jefferson Medical College, 1867. Appointed, 1882. Visiting Surgeon to the Jefferson Medical College; Clinical Professor of Surgery in the Jefferson Medical College.

LEWIS W. STEINBACH, M.D., 1309 North Broad Street. Graduate of Jefferson Medical College, 1880. Appointed, 1885. Surgeon to the Jewish Hospital; Professor of Surgery in the Philadelphia Polyclinic.

JOHN H. MUSSER, M.D., 1927 Chestnut Street. Graduate of University of Pennsylvania, 1877. Appointed, 1885. Assistant Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Presbyterian Hospital; Consulting Physician to the Woman's Hospital, and to the West Philadelphia Hospital for Women; President of the Pathological Society of Philadelphia.

HENRY W. STELWAGON, M.D., 223 South Seventeenth Street. Graduate of University of Pennsylvania, 1875. Appointed, 1887. Clinical Professor of Dermatology in the Jefferson Medical College, and in the Woman's Medical College; Physician to the Skin Department of the Northern Dispensary, and of the Howard Hospital.

FRANCIS X. DERCUM, M.D., 1719 Walnut Street. Graduate of University of Pennsylvania, 1877. Appointed, 1887. Clinical Professor of Diseases of the Nervous System in the Jefferson Medical College; Visiting Physician to St. Clement's Hospital for Epileptics; Consulting Neurologist to St. Agnes' Hospital; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases.

G. E. DE SCHWEINITZ, M.D., 1401 Locust Street. Graduate of University of Pennsylvania, 1881. Appointed, 1887. Professor of Ophthalmology in Philadelphia Polyclinic and School for Graduates; Clinical Professor of Ophthalmology in Jefferson Medical College; Ophthalmic Surgeon to the Children's Hospital, to the Orthopedic Hospital and Infirmary for Nervous Diseases, and to the Methodist Hospital; Ophthalmologist to the Church Home, Angora, and to the Home of the Merciful Saviour for Crippled Children; Consulting Ophthalmologist to the Chester County Hospital, to the Bryn Mawr Hospital, and to the Hospital for Epileptics and Dispensary of St. Clement's Church.

BARTON COOKE HIRST, M.D., 1821 Spruce Street. Graduate of University of of Pennsylvania, 1883. Appointed, 1887. Professor of Obstetrics in the University of Pennsylvania; Obstetrician to the University and Maternity Hospitals; Gynecologist to the Orthopedic Hospital.

FREDERICK P. HENRY, M.D., 1635 Locust Street. Graduate of College of Physicians and Surgeons, New York, 1868. Appointed, 1887. Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania; Physician to the Home for Consumptives at Chestnut Hill.

EDWARD MARTIN, M.D., 415 South Fifteenth Street. Graduate of University of Pennsylvania, 1883. Appointed, 1888; served until 1889; re-elected, 1892. Served as Surgical Registrar from 1885 to 1888. Clinical Professor of Genito-Urinary Diseases in the University of Pennsylvania.

JAMES HENDRIE LLOYD, M.D., 3910 Walnut Street. Graduate of University of Pennsylvania, 1878. Appointed, 1888; served until December, 1889; reappointed, 1890. Physician to the Methodist Episcopal Hospital; Physician to the Home for Crippled Children; Neurologist to the Pennsylvania Training School for Feeble-Minded Children at Elwyn; Consulting Neurologist to the State Hospital for the Chronic Insane at Wernersville.

EDWARD P. DAVIS, M.D., 250 South Twenty-first Street. Graduate of Rush Medical College, Chicago, 1882. Appointed, 1888. Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Clinical Professor of Obstetrics in the Jefferson Medical College.

WILLIAM E. HUGHES, M.D., 3945 Chestnut Street. Graduate of the University of Pennsylvania, 1880. Appointed, 1889. Professor of Clinical Medicine in the Medico-Chirurgical College; Visiting Physician to the Medico-Chirurgical Hospital; Pathologist to the Presbyterian Hospital.

SOLOMON SOLIS-COHEN, M.D., 1525 Walnut Street. Graduate of Jefferson Medical College, 1883. Appointed, 1889. Professor of Clinical Medicine and Applied Therapeutics in the Philadelphia Polyclinic; Lecturer on Clinical Medicine in the Jefferson Medical College; Consulting Physician to the Jewish Hospital; Physician to the Rush Hospital; Consulting Laryngologist to the Pennsylvania Institution for Feeble-Minded Children.

ORVILLE HORWITZ, M.D., 1721 Walnut Street. Graduate of Jefferson Medical College, 1883. Appointed, 1889. Clinical Professor of Genito-Urinary Diseases, Jefferson Medical College; Professor of Genito-Urinary Surgery, Philadelphia Polyclinic.

ERNEST LAPLACE, M.D., 1828 South Rittenhouse Square. Graduate of University of Louisiana, 1884. Appointed, 1889. Professor of Clinical Surgery in the Medico-Chirurgical College.

GEORGE MORLEY MARSHALL, M.D., 1819 Spruce Street. Graduate of University of Pennsylvania, 1886. Appointed, 1890. Attending Physician to St. Joseph's Hospital and Chief of its Throat Dispensary.

GEORGE I. MCKELWAY, M.D., 114 South Eighteenth Street. Graduate of University of Pennsylvania, 1889. Appointed, 1890.

RICHARD C. NORRIS, M.D., 500 North Twentieth Street. Graduate of University of Pennsylvania, 1887. Appointed Registrar, 1890; Obstetrical Staff, 1894; Lecturer on Clinical and Operative Obstetrics, University of Pennsylvania; Physician in Charge, Preston Retreat; Gynecologist to Methodist Episcopal Hospital; Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital for Women and Children.

J. CHALMERS DA COSTA, M.D., 1629 Locust Street. Graduate of Jefferson Medical College, 1885. Appointed Registrar, 1890. Appointed on Surgical Staff, 1895. Demonstrator of Surgery, Jefferson Medical College; Chief Assistant Surgeon, Jefferson Medical College Hospital.

JULIUS L. SALINGER, M.D., 1510 North Eighth Street. Graduate of Jefferson Medical College, 1886. Appointed, 1892. Lecturer on Renal Diseases, Jefferson Medical College; Chief of the Medical Clinic, Jefferson Medical College Hospital.

AUGUSTUS A. ESHNER, M.D., 224 South Sixteenth Street. Graduate of Jefferson Medical College, 1888. Appointed Registrar, 1891, and Visiting Physician, 1896; Professor of Clinical Medicine in the Philadelphia Polyclinic; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases.

ALFRED STENGEL, M.D., 1811 Spruce Street. Graduate of the University of Pennsylvania, 1889. Appointed Medical Registrar, 1892, and Visiting Physician, 1895. Formerly Physician to the Howard Hospital, also Pathologist to the German Hospital; now Physician to the Children's Hospital; Professor of Clinical Medicine in the University of Pennsylvania.

JOHN M. FISHER, M.D., 1527 Wallace Street. Graduate of Jefferson Medical College, 1884. Appointed, 1894. Chief of the Department of the Diseases of Women, and Demonstrator of Gynecology in the Jefferson Medical College Hospital.

THOMAS G. ASHTON, M.D., 1533 Pine Street. Graduate of Jefferson Medical College, 1888. Appointed, 1894. Chief of the Out-Patient Medical Department of the Jefferson Medical College Hospital; Demonstrator of Clinical Medicine, Jefferson Medical College; Visiting Physician to St. Mary's Hospital.

CHARLES A. OLIVER, M.D., 1507 Locust Street. Graduate of the University of Pennsylvania, 1876. Appointed, 1894. Attending Surgeon to the Will's Eye Hospital; Ophthalmic Surgeon to Presbyterian Hospital; Consulting Ophthalmic Surgeon to St. Agnes', St. Mary's, St. Timothy's, and Maternity Hospitals; Consulting Ophthalmologist to State Hospital for Chronic Insane of Pennsylvania; Ophthalmologist to the Friends' Asylum for the Insane, Frankford.

W. FRANK HAEHNLEN, M.D., 1616 Walnut Street. Graduate of the University of Pennsylvania, 1882. Appointed, 1895. Professor of Obstetrics, Medico-Chirurgical College; Obstetrician to Medico-Chirurgical and Maternity Hospitals; Medical Director and Gynecologist, Samaritan Hospital.

ALFRED C. WOOD, M.D., 214 South Fifteenth Street. Graduate of the University of Pennsylvania, 1888. Appointed, 1895. Assistant Surgeon to the University Hospital; Instructor in Clinical Surgery in the University of Pennsylvania.

ELIZABETH L. PECK, M.D., 819 North Fortieth Street. Graduate of Woman's Medical College of Pennsylvania, 1885. Appointed, 1895. Visiting Physician, West Philadelphia Hospital for Women; Instructor in Materia Medica and Therapeutics, Woman's Medical College of Pennsylvania.

CHARLES W. BURR, M.D., 1327 Spruce Street. Graduate of the University of Pennsylvania, 1886. Appointed, 1896. Formerly Professor of Mental and Nervous Diseases in the Medico-Chirurgical College, and the Philadelphia Polyclinic.

W. M. L. COPLIN, M.D., 1629 South Broad Street. Graduate of Jefferson Medical College, 1886. Appointed, 1892. Resigned, 1895. Reappointed, November, 1896. Professor of Pathology in the Jefferson Medical College.

CHARLES H. FRAZIER, M.D., 133 South Eighteenth Street. Graduate of University of Pennsylvania, 1892. Appointed, 1898. Professor of Clinical Surgery in the University Hospital; Surgeon to the University and Howard Hospitals; Surgeon to the Home for Crippled Children.

JOHN B. SHOBER, M.D., 1731 Pine Street. Graduate of the University of Pennsylvania, 1885. Appointed, December, 1896. Gynecologist to the Howard Hospital; Assistant Surgeon to the Gynecean Hospital; Visiting Physician to the Bar Harbor Hospital, Maine.

L. NAPOLEON BOSTON, M.D., 1524 Snyder Avenue. Graduate of Medico-Chirurgical College. Appointed, 1898. Bacteriologist, Pennsylvania Hospital; Demonstrator in Charge of Clinical Laboratory, Medico-Chirurgical College.

CHARLES L. LEONARD, M.D., 1930 Chestnut Street. Graduate of the University of Pennsylvania, 1889. Appointed, 1899.

H. AUGUSTUS WILSON, M.D., 1611 Spruce Street. Graduate of Jefferson Medical College, 1879. Appointed, January, 1900. Clinical Professor of Orthopedic Surgery, Jefferson Medical College; Emeritus Professor of Orthopedic Surgery, Philadelphia Polyclinic; Consulting Orthopedic Surgeon to the Kensington Hospital for Women; Consulting Orthopedic Surgeon, Philadelphia Lying-in Hospital.

HOWARD F. HANSELL, M.D., 254 South Sixteenth Street. Graduate of Jefferson Medical College, 1879. Appointed, March, 1900. Clinical Professor of Opthalmology, Jefferson Medical College; Professor of Diseases of the Eye, Philadelphia Polyclinic, and College for Graduates in Medicine; Ophthalmic Surgeon to the Frederick Douglass Memorial and the Chester County Hospital.

HERMAN B. ALLYN, M.D., 501 South Forty-second Street. Graduate of the University of Pennsylvania, 1885. Appointed Medical Registrar, 1898, and Visiting Physician, June, 1900. Instructor in Physical Diagnosis in the University of Pennsylvania; Visiting Physician, St. Joseph's Hospital.

DAVID RIESMAN, M.D., 326 South Sixteenth Street. Graduate of the University of Pennsylvania, 1892. Appointed Visiting Physician, June, 1900. Professor of Clinical Medicine, Philadelphia Polyclinic; Instructor in Clinical Medicine, University of Pennsylvania; Consulting Physician to the Jewish Hospital; Neurologist to the Northern Infirmary.

MILTON B. HARTZELL, M.D., 3644 Chestnut Street. Graduate of Jefferson Medical College, 1877. Appointed, July, 1900. Instructor of Dermatology in the University of Pennsylvania; Dermatologist to the Methodist Episcopal Hospital.

E. S. GANS, M.D., 711 North Franklin Street. Graduate of Jefferson Medical College. Appointed, 1900. Lecturer on Dermatology in the Medico-Chirurgical College.

JOSEPH MCFARLAND, M.D., 442 W. Stafford Street, Germantown, Phila. Graduate of the University of Pennsylvania, 1885. Appointed, October, 1900. Professor of Pathology, Medico-Chirurgical College.

SIMON FLEXNER, M.D., 218 South Fifteenth Street. Graduate of University of Louisville, Ky. Appointed, October, 1900. Professor of Pathology in the University of Pennsylvania.

F. SAVERY PEARCE, M.D., 1407 Locust Street. Graduate of the University of Pennsylvania, 1891. Appointed, January, 1901. Professor of Diseases of Nervous System, Medico-Chirurgical College.

WILLIAM C. SPILLER, M.D., 4409 Pine Street. Graduate of the University of Pennsylvania, 1892. Appointed, January, 1901. Assistant Professor of Diseases of Nervous System, University of Pennsylvania.

CHARLES S. POTTS, M.D., 1726 Chestnut Street. Graduate of the University of Pennsylvania, 1885. Appointed, January, 1901.

JOHN W. CROSKEY, M.D., 1831 Chestnut Street. Graduate of the Medico-Chirurgical College, 1889. Appointed, January, 1901.

EDWARD B. GLEASON, M.D., 41 South Nineteenth Street. Graduate of the University of Pennsylvania, 1880. Appointed, January, 1901.

CHARLES P. GRAYSON, M.D., 251 South Sixteenth Street. Graduate of Jefferson Medical College, 1880. Appointed, January, 1901.

JAMES P. MANN, M.D., 1234 Spring Garden Street. Graduate of Jefferson Medical College, 1887. Appointed, January, 1901.

GWILYN G. DAVIS, M.D., 255 South Sixteenth Street. Graduate of the University of Pennsylvania, 1879. Appointed, January, 1901.

WILLIAM C. HOLLOPETER, M.D., 1428 North Broad Street. Graduate of the University of Pennsylvania, 1877. Appointed, January, 1901. Professor of Diseases of Children, Medico-Chirurgical College.

EDWARD E. GRAHAM, M.D., 1713 Spruce Street. Graduate of Jefferson Medical College, 1887. Appointed, January, 1901. Clinical Professor of Diseases of Children, Jefferson Medical College.

J. P. CROZER GRIFFITH, M.D., 123 South Eighteenth Street. Graduate of the University of Pennsylvania, 1881. Appointed, January, 1901.

J. MADISON TAYLOR, M.D., 1504 Pine Street. Graduate of the University of Pennsylvania, 1878. Appointed, January, 1901.

R. H. NONES, D.S., 1708 Chestnut Street. Graduate of Philadelphia Dental College, 1885. Appointed Dental Surgeon, January, 1901.

M. H. CRYER, D.S., 504 Crozier Building. Graduate of Philadelphia Dental College, 1876. Appointed Dental Surgeon, January, 1901.

I. NORMAN BROOMELL, D.S., 901 Crozier Building. Graduate of Pennsylvania College of Dental Surgery, 1879. Appointed Dental Surgeon, January, 1901.

THOMAS C. STELLWAGON, JR., D.S., 501 Hale Building. Graduate of Philadelphia Dental College, 1897. Appointed Dental Surgeon, January, 1901.

B. FRANKLIN STAHL, M.D., 1502 Arch Street. Graduate of the University of Pennsylvania, 1887. Appointed Registrar, 1896. Physician to St. Agnes' Hospital.

W. A. NEWMAN DORLAND, M.D., 120 South Seventeenth Street. Graduate of the University of Pennsylvania, 1886. Appointed, January, 1898.

JOSEPH SAILOR, M.D., 330 South Sixteenth Street. Graduate of the University of Pennsylvania, 1891. Appointed Registrar, July, 1900.

WILLIAM PICKETT, M.D., 1508 North Fourth Street. Graduate of Jefferson Medical College, 1895. Appointed Registrar, August, 1900.

R. D. NEWTON, M.D., 2705 Oxford Street. Graduate of Medico-Chirurgical College, 1896. Appointed Registrar, October, 1900.

JAMES H. MCKEE, M.D., 1519 Poplar Street. Graduate of the University of Pennsylvania, 1892. Appointed Registrar, 1901.

OFFICIAL CHANGES.

WHEN the third volume of the PHILADELPHIA HOSPITAL REPORTS was printed in 1896, the Department of Charities and Correction was composed of the Hon. Charles F. Warwick, Mayor, *ex-officio;* William H. Lambert, President; William D. Gardner, Treasurer; Alfred Moore, James A. Walk, M.D., and John Shallcross.

In April, 1897, the term of five years (for which the directors are appointed) having expired, the Mayor reappointed William H. Lambert President, and Alfred Moore and William D. Gardner with Joseph H. Mann and Henry B. Gross as Directors.

In January, 1899, Joseph H. Mann (after a lingering illness) died. He had been a very active member of the Board, and a suitable minute was passed attesting to his worth as a citizen, his integrity as a merchant, and his usefulness and Christian spirit as a director. The Mayor, in March, 1899, appointed as the successor of Mr. Mann, Dr. Joseph F. Neff, who, from 1884 to 1887, had been one of the Visiting Physicians of the Philadelphia Hospital. In February, 1899, Henry B. Gross resigned. In April, 1899, William H. Lambert, President, resigned. The administration of Mr. Lambert was one of the most satisfactory and successful in the history of the institution. His resignation was an acknowledged loss in all respects.

The Mayor-elect, Hon. Samuel H. Ashbridge, appointed Albert H. Dingee and Dr. Caleb S. Middleton as Directors to fill the vacancies caused by the resignations of President Lambert and Mr. Gross, and named William D. Gardner President *pro tem.*

In May, 1900, Alfred Moore resigned to accept the appointment as Director of the Board of City Trusts. The Mayor appointed Dr. John V. Shoemaker to fill the vacancy caused by Mr. Moore's resignation, and soon after named Dr. Shoemaker President of the Department, in place of Mr. Gardner, who had been the acting President since the resignation of Mr. Lambert in April, 1899.

August 2, 1900, Mr. William D. Gardner (Treasurer, and, for fifteen months, the acting President) died after a short illness. The Board

adopted the following resolution: "In the death of our late colleague, William D. Gardner, we have lost a sincere friend and the Board a faithful member. In his long connection with the department he attended strictly to the various duties he was called upon to perform, retaining an active interest to the last. Kind to those who sought his advice, courteous to those connected with him officially, and genial with his associates."

The Mayor appointed Dr. Ellwood R. Kirby as Director to fill the vacancy caused by the death of Mr. Gardner.

In September, 1900, Dr. Joseph F. Neff resigned, and the Mayor named William J. McLaughlin as Director to fill the vacancy caused by the resignation of Dr. Neff.

In June, 1900, William M. Geary was appointed Superintendent in place of Charles Lawrence, resigned.

In November, 1900, the functions of the Superintendent of the Institution were extended to include the duties and authority of the Bureau of Charities, under the title of Superintendent of Bureau of Charities, Mr. Geary being continued in the new position.